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An Address.[1]

TIMES AND THE DOCTOR.[1]

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Retiring President of the Tasmanian Branch of the British Medical Association.

WHILST the preparation and delivery of an address are the most formidable of the President's duties, there are several rewards. One is in the study one makes of the published efforts of one's predecessors. It is a comfort to find that the choice of a subject has been a difficulty to so many more distinguished speakers than myself. As long ago as 1899 Duncan, addressing the Medical Society of Victoria, observed that past speakers had used almost every possible subject. Nevertheless he was able to develop his theme, in the manner of the day, for a further 26 pages of the *Colonial Medical Journal*.

[1] Delivered at the annual meeting of the Tasmanian Branch of the British Medical Association on March 12, 1960.

This address is divided into two sections; the first comprises some reflections of a general nature, and the second, observations on my particular branch of the practice of medicine as seen in Tasmania.

In our invaluable Book of Rules, the only precise instructions to the president, other than on the conduct of meetings, are that he shall "generally superintend the conduct of the affairs of the Branch". In recent years there has been a strong feeling of democratic principle in successive Branch Councils and their presidents. The past year, I feel, has been no exception. Malcontents may note that your Council has endeavoured at all times to interpret the wishes of the majority.

Publicity.

From time to time efforts are made by enthusiasts to organize some regular medium of publicity on medical matters. Fortunately, in my opinion, these efforts always fail. To find the right person to produce such work is difficult, and to maintain a regular output is impossible in a small community.

The general public remains suspicious of organized medicine, as well it may be. It goes to a doctor chiefly

when driven by injury or ill health, and it does not, by and large, have any true interest in medicine when in good health. To produce regular reminders that there is an organized profession living by means of the ill health of the unlucky seems to me to be tactless in the extreme.

Professional reporting of medical news is in another category. However inaccurate or melodramatic the item may be, here there is no vested interest. Because it is written by professionals, the news is acceptable and, let us confess, often of as much interest to the medical as to the lay reader. The fact remains that, with little publicity from itself, the members of the medical profession, as individuals, enjoy a standing in the community as high as they have known for many years. For this we can thank our predecessors in practice. It is a standing which has been won by the exhibition of integrity and selflessness in the consulting-room and in the private home. It is important in the struggle of private medical practice against socialism, no less than for our personal welfare, that these standards be maintained.

Ethics.

Of a reference to ethics, few presidential addresses are free.

Since the last war, there has been in Australia ample medical work for the willing. This brings a much reduced need for scrupulous dealing between practitioners over straying patients. Often the original doctor is pleased to see the last of such a patient.

This situation does not diminish the obligation of the practitioner to the patients in his practice. I wish to refer specifically to the habit in this State of a doctor leaving his practice without making an arrangement for his work to be done in his absence. Such conduct prejudices the cause of private practice in the public's mind.

To the newcomer I recommend still the courtesy call, at least on one's neighbouring competitors, and on a selection of the eminent of the town. The feelings of such colleagues, some of whose patients may shortly be yours, may be expressed as "better the devil you know . . .". Your own turn will come only too soon.

Leadership in Medical Life.

"To every action there is an equal and opposite reaction." This elementary law of physics would seem to sum up the efforts of the majority of medical men who essay to organize their professional colleagues towards better things. I have witnessed several times the frustration that awaits the medical Machiavelli in this State. Fortunately for most of us, our power complexes are more than sublimated by the influence we are obliged to exert on the lives of our patients. Medical leadership there must be; but unless the personality is outstanding, the intending benefactor should proceed with a maximum of discreet inertia, in order to delay the onset of the inevitable reaction for as long as possible.

Medical School.

Perhaps the most stimulating event in the medical year just ended has been the movement towards a medical school for Tasmania. The case has been admirably presented in its report by the Parliamentary Select Committee, the medical members of which are deserving of our thanks and commendation. At the present time several avenues are being explored. I am sorry not to have been able to come to this meeting with a report of success.

Obstetrics in Tasmania.

To general obstetrical history I have but one reference to make. When it appeared that a natural termination to the labour of Jane Seymour could not be expected, Henry VIII was asked whether he willed that the mother's or the child's life be spared. He replied: "Save the child by all means, for I shall be able to get mothers enough."

One of the most interesting accounts of obstetrical happenings in early Tasmanian history was published in THE MEDICAL JOURNAL OF AUSTRALIA in 1949 by Dr. W. E. L. H. Crowther. The delivery, which took place at Kempton, ended in a double tragedy. It is relevant to my theme because the labour was prolonged for over 60 hours, during most of which time the doctor was at the bedside of the patient.

Much of progress in obstetrics is concerned with shortening of the time factor, as some of the figures given later will show. The change in *tempo* and in thought is typified by the title of a review published by Corbett in *The Lancet* in 1958. This was called "The Unforgiving Minute in Obstetrics".

For the data used in the following survey of current conditions of obstetrical practice in Tasmania, two sources have been used. The first has been provided jointly by the Registrar-General and the Director-General of Health Services, in the shape of their records of maternal deaths since 1950. For the courtesy and cooperation of these gentlemen and their staffs I am most grateful. For the second source of data, records from the Queen Victoria Hospital, Launceston, have been used. As many of you know, the interesting feature of this hospital is that the medical care is in the hands of a small number of doctors, nearly all of whom have special training or experience in the subject. I am grateful to my colleagues for permission to use records of their cases.

Maternal Mortality.

In 1954 a standing committee was appointed to consider each maternal death in Tasmania. As a result, many more details are available for the study of such cases since that time, and the great value of the activities of the committee is unquestionable.

For the perior 1950-1958, 52 maternal deaths have been traced. These included 12 deaths in early pregnancy due to the following causes: sepsis following abortion; embolism; ectopic pregnancy; nephritis. In this period, 71,253 live births, and 1163 stillbirths were registered. The maternal mortality rate is therefore 0·74 per 1000 live births.

This figure compares favourably with the New South Wales rate of 0·82, reported by the Special Medical Committee (1960). However, it has been found in Tasmania that some maternal deaths are untraceable if no indications of pregnancy are given amongst the terms used on the death certificate. Hence this figure may be lower than the true rate.

In Scotland, in 1957, only one maternal death occurred in each 2000 deliveries, a rate of 0·5 per 1000 births.

Causes of Death.

Excluding the deaths in early pregnancy, analysis of the causes of death gives the following results: post-partum hæmorrhage, seven cases; pulmonary embolism, seven cases; toxæmia, six cases; cardiac failure, five cases; anæsthesia, two cases; accidental hæmorrhage, two cases; malignant melanoma, hæmorrhage (placenta prævia), hæmorrhage (cerebral), puerperal insanity (suicidal), pneumonia (staphylococcal), blood transfusion, rupture of the uterus, sepsis, chronic nephritis, infective hepatitis, and retained placenta and shock, one case each; total, 40.

I should like to comment on some of these causes in detail.

Post-Partum Hæmorrhage.—Of post-partum hæmorrhage I would say that there can be no easiness of the conscience for those who fail to use an oxytocic, preferably ergometrine, at the conclusion of the second stage of labour.

In the first 100 normal deliveries at the Queen Victoria Hospital, Launceston, in 1949, before the use of such a method was adopted, the average blood loss was 13·8 oz. There were five blood transfusions given in cases in which the losses recorded were up to 80 oz. In the first 100 normal deliveries in 1959, in which ergometrine was administered intravenously after the birth of the

baby, the average blood loss was 5·6 oz., the highest loss recorded was 35 oz., and no blood transfusions were required. Nevertheless, hæmorrhage does still occur, and no obstetrical centre should be without its organization for the prompt giving of a blood transfusion. Of other methods of treatment, perhaps the pack is the most helpful; but the times which prompted Playfair, in 1860, to state that "the puerperal uterus when correctly packed will hold two ball dresses" have all but passed.

Pulmonary Embolism.—Of the seven fatalities from pulmonary embolism, three were uncomplicated, two patients had mitral valve lesions, one patient was noted to have thrombo-phlebitis of a calf vein, and one death followed Cæsarean section for preeclampsia.

Toxæmia.—Of the six patients, four had developed eclampsia before they died. The prophylactic treatment of preeclampsia is well carried out in the chief centres in this State.

Cardiac Failure.—Of the five cases of cardiac failure traced, in none do the certified causes of death reveal the ætiology of the condition. Prevention of tragedy in most of such cases lies in careful assessment and the making of a decision in the first trimester of the pregnancy.

Anæsthetics.—Both of the anæsthetic deaths occurred in the phase of induction. One was due to "Pentothal" sensitivity. Neither was related to the bugbear of inhalation of vomitus. Our trend is away from general anæsthesia. In 200 consecutive vaginal deliveries in 1940, ether, chloroform or a mixture of the two was used in every case in which an anæsthetic was required. In 1959, in 200 consecutive vaginal deliveries in the practices of two specialists, local anæsthesia, with or without "Trilene" as an adjunct, was used in 51 deliveries (25%). Ether was administered 112 times. Chloroform was not used. In 27 cases "Trilene" was the only anæsthetic agent used. Ten patients received no anæsthetic, owing either to the speed of the delivery or to the practice of "natural childbirth".

It has been interesting to follow the reaction of patients as the news of delivery with local anæsthesia has filtered through the community. Specific requests for general anæsthesia are few, but a number of patients ask that they may be awake for their delivery. The final choice should be made by the obstetrician.

For the future, assessments will be more accurate if the death certificates include an indication that the case was obstetrical, and of a term to indicate the ætiology when possible.

The Third Stage of Labour.

Mention has been made of the revolution brought to the management of the third stage of labour by the elimination of the menace of post-partum hæmorrhage by the intravenous administration of ergometrine. One who must view this advance with satisfaction is W. Keverall McIntyre, whose pioneer work with the Mojon-Gabaston cord injection for the control of post-partum hæmorrhage was arousing interest 20 years ago.

Another revolution, most welcome to patient and attendant, which has accompanied the ergometrine injection, has been the shortening of the duration of this stage of labour. In 1940, the average duration of the third stage in 200 consecutive vaginal deliveries was 20·75 minutes. In 1959, the average for 200 such cases was 7·1 minutes. For the patient, this shorter time reduces exposure and the discomfort of lying in a bed that is generally damp. There is a corresponding decrease in the amount of shock sustained. For the obstetrician, the time saved is important. Calculated at 13 minutes a case, in a practice of 400 cases *per annum* (a figure not uncommon for Tasmanian specialist obstetricians), the saving is 86 hours, or 100 minutes a week, or 14 minutes a day.

The belief is growing that the shorter the time that the placenta remains *in utero*, the less likelihood there is that shock will develop. When the patient is delivered under general anæsthesia, the placenta should be delivered during the same anæsthesia, after the administration of the oxytocic, if necessary by immediate manual removal. Such patients recover consciousness with no ill effect from the extra manipulation. There is no shock, and the extra blood lost is insignificant.

This time saving, together with the reduction of the number of complications from hæmorrhage and shock, has facilitated the concentration of deliveries into the hands of specialists. Much may be said, in debate, about the "occasional obstetrician", the value of the family doctor, and whether the midwifery should be subtracted from his practice. There is no doubt in my mind that the present methods make for a smooth service, with good results in terms of successful births, and a minimum of maternal morbidity and mortality.

Total Labour Times.

The times from onset of labour to delivery for primigravidæ and multigravidæ in the foregoing series were as follows: (i) 1940: primiparæ, 24 hours; multiparæ, 11 hours; (ii) 1959: primiparæ, 16·9 hours; multiparæ, 10·1 hours.

If the difference in the durations of the first stages in the primiparæ groups is significant, it is due probably to the change in routine sedation from morphine to pethidine. The latter facilitates dilatation of the cervix, although its analgesic effect is variable.

The Perineum.

As an advocate of local anæsthesia, I am asked not infrequently what effect this has on the episiotomy rate. The figures taken from 200 consecutive vaginal deliveries in 1940 and contrasted with those of 1959 are as shown in Table I.

TABLE I.

	1940.	1959.
Perineum intact	60%	52%
Episiotomy	11%	38%
Perineum lacerated	28%	9%

The decrease in the intact perineum rate is thus 8%.

The change to episiotomy from trying for the preservation of the perineum is striking. It is no longer thought that the intact perineum is a hallmark of success. Pupil nurses may now dare to ask for an episiotomy to be done. Patients have come to believe that stitches are respectable, and possibly an insurance against "falling of the womb", about which their elder sisters delight to tell them.

Husbands.

With the advent of New Australians, there is a demand for admission of husbands to labour wards and delivery rooms. Many of us have deferred this problem with the excuse that our hospital layouts are unsuitable, or that nursing problems of mass deliveries in hospital do not permit the entry of these intruders. What is to be our attitude when building time comes? Should these people be educated out of their traditional ways?

Few Australian-born husbands seek these adventures, and of the few, some have had to be assisted to the fresh air.

There is no final answer, and we should remain ready to compromise.

Natural Childbirth.

Allied to the question of husbands at the delivery is the current, although perhaps waning, vogue of so-called natural childbirth. Here again there is no final answer. What is one primipara's meat is another multipara's poison. The latter class of patient is inclined to ask for all the assistance with analgesia and anæsthesia that modern methods can give her.

The true "natural delivery" by an exhilarated young primipara is a fine sight. Some of these patients may not ever have heard of Grantly Dick Read, or have received any special instruction. Others benefit greatly by their training in this technique.

It behoves the obstetrician to adapt his technique to all such types, remembering that mothers are no more all alike than are their babies—an error of thought into which our infant welfare clinics are prone to fall.

On the scientific side I quote Helen Rodway (1958), who reported a series of 2700 primiparæ from the Thorpe Coombe Hospital. One group was given instruction in relaxation, exercises and natural childbirth by trained physiotherapists. The second group received no preparation at all. There was no difference between the average durations of either the first or second stages of labour in the two groups.

The Registration of Births Form.

Finally I would make reference to the birth registration form, which was introduced to Tasmania early in 1959. After a lukewarm reception, the initial trial period is now concluded. It was only on attending the conference held to discuss the future of the form at Hobart in January of this year that it became clear to me what the objectives were. It may be of value if these are outlined here.

The inspiration for the trial of this form comes from the World Health Organization through the National Health and Medical Research Council of Australia. Tasmania was chosen for the trial, because schemes for other States had been delayed by the difficulties caused by their larger populations. The figures now being processed by the Commonwealth Statistician's Department will be made available to all the practitioners of this State. They are expected to be a guide to research workers, statisticians and university teachers.

I suggest that these figures are ours, to be made use of in the preparation of scientific papers, and in contributions to clinical meetings. It is seldom that such an opportunity is presented to clinicians in this State to report on something which should prove to be of national interest.

Conclusion.

Thus, from the difficulties of the last century, we find ourselves in 1960 in the front line of a World Health Organization project. Yet no matter to what level of simplicity the normal labour is reduced, the lure of obstetrics will remain:

> Age cannot wither her, nor custom stale
> Her infinite variety.

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PIGMENTATION OF THE NAIL-BEDS, PALATE AND SKIN OCCURRING DURING MALARIAL SUPPRESSIVE THERAPY WITH "CAMOQUIN".

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IN a previous communication (Campbell, 1959) the occurrence of a bluish-grey pigmentation of the face, nail-beds and palate during long-term malarial suppressive therapy with "Camoquin" (amodiaquine hydrochloride) was noted. In order to determine the approximate incidence of this pigmentation, an investigation was undertaken in which 250 European adults of both sexes, comprising hospital staff and hospital in-patients and out-patients, were examined. One hundred of these people were taking "Camoquin" relatively regularly as a malarial suppressive, usually in a dose of 0·4 or 0·6 gramme of "Camoquin" base per week.

At the same time, 714 Port Moresby European school and pre-school children of both sexes, whose ages ranged from three to 15 years, were examined to see whether this pigmentation occurred in children and to determine its approximate incidence. Of these children, 291 were taking "Camoquin" regularly in appropriate age dosages each week.

RESULTS OF INVESTIGATION.

Description of the Pigmentation.

The pigmentation usually affects four principal sites: (i) one or more of the nail-beds of the toes on one or both feet; (ii) the mucous membrane of the hard palate; (iii) one or more of the nail-beds of the fingers on one or both hands; (iv) the skin of the face and neck.

The relative distribution of the pigmentation among these four sites is quite variable, and appears to bear no relationship to the duration of the suppressive course of "Camoquin", except that pigmentation of the face has been noticed only after more than two years of suppressive "Camoquin" therapy. Only one site, two or more sites, or all four of the principal sites may be involved.

Very rarely, in addition the mucous membrane of the lips, cheeks or gums or the skin of other parts of the body may be involved.

Toes.

The pigmentation on the toes is of a dark-grey to black colour, and commences as small irregular macules in the middle third of the nail-bed. It may eventually form a distinct dark band across the middle third of the nail-bed, which is, in the case of the great toe, about 3 to 4 mm. broad. The great toe is the toe most commonly involved, and in addition one or all of the other toes may be involved in a similar fashion. The distribution of the pigmentation is sometimes symmetrical in both feet (Figure I).

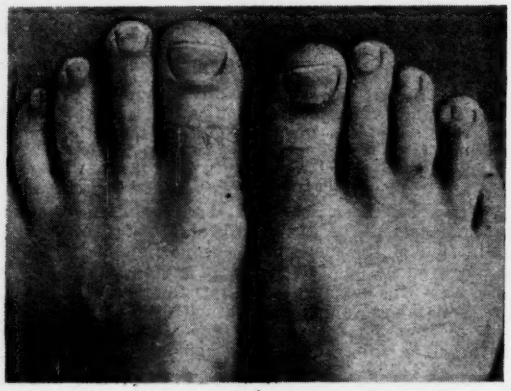

FIGURE I.

Showing pigmentation of the middle third of the subungual tissues of the toes in a man who had been taking "Camoquin" as a malarial suppressive for five years.

Palate.

The pigmentation on the palate is of a bluish-grey to almost black colour and affects the mucous membrane of the hard palate (Figure II[1]). It commences as irregular patches or streaks of pigment on one or both sides of the mid-line of the arch of the palate. These may eventually coalesce to form a very dark band, half an inch wide on either side of the mid-line, which commences one inch behind the incisor teeth and extends back to the posterior border of the hard palate. Sometimes a fine line

[1] For Figures II, III and IV, see art-paper supplement.

of pigment extends laterally and backwards from the main pigment mass to the last molar tooth on either side.

Fingers.

The pigmentation on the fingers is of a similar colour to that affecting the toes, and commences as one or more small, irregularly shaped macules in the proximal two-thirds of the nail-bed. It may eventually form a dark pigmented area which usually occupies the proximal half to two-thirds of the nail-bed, but it may form a mid-third band. From one to 10 digits may be involved.

Face.

On the face the colour of the pigmentation is variable; it may be brown, bluish-grey or grey. The pigmentation may occur as a small area of diffusely scattered pigment, or as one or two or more isolated pigmented areas, or as a diffuse pigmentation of the whole or part of the face. The tip of the nose and the alæ nasæ are commonly involved, and may look as if they have been smudged with an ink-stained finger (Figure III). Sometimes a triangular or linear area of pigmentation about 12 mm. long has been present on the dorsum or root of the nose. Irregularly-shaped pigmented macules have occurred on other parts of the face and lips. Some people, who have been taking "Camoquin" as a suppressive for over three years, have developed a diffuse grey or bluish-grey pigmentation of the whole face and neck (Figure IV). Superimposed on this diffuse pigmentation, and scattered over the face and neck, there may be numerous grey or brown irregularly shaped macules up to 1 cm. in diameter.

Less Common Sites.

One person in the present investigation had a bluish-grey macule on the gum margin, and another had a greyish pigmentation of the skin of the dorsum of one toe.

In addition, two people with facial and nail-bed pigmentation due to "Camoquin", who are not included in the 100 people reported here, have been seen. One of these people had an extensive bluish-grey mottling of the mucous membranes of the cheeks and lips and small bluish-grey macules on edentulous gums. The other person had an isolated bluish-grey macule on one knee.

Incidence of Pigmentation.

Adults.

One hundred adults (65 males, 35 females) were taking "Camoquin" regularly for periods of time varying from five weeks to over six years; 40 of them (29 males, 11 females) showed evidence of pigmentation at one or more of the four principal sites. The commonest site affected was the nail-bed of the great toe (29 cases). Next in frequency was the mucous membrane of the hard palate (27 cases), then came the nail-beds of the fingers (18 cases), and the least commonly affected area of all was the skin of the face (eight subjects, of whom three had diffuse pigmentation of the face and neck).

The incidence of the pigmentation increased with the duration of suppressive therapy, as is shown in Table I. The shortest course required to produce the pigmentation was nine months.

Of the 50 adults who had been taking "Camoquin" regularly for two or more years, pigmentation (all types) was found in 33 (23 of 34 males, 10 of 16 females). The pigmentation was generally less dense in the females examined, and its incidence did not appear to be significantly different on a dose of 0·4 or 0·6 gramme of "Camoquin" base per week. Once pigmentation appeared at a particular site, people have noted that it tended to become denser as the suppressive course was continued.

Five adults with pigmentation have been followed for periods which vary from six to 14 months after the cessation of "Camoquin" therapy. The isolated patches of pigment on the face take approximately six months to disappear completely. The pigmentation of the nail-beds may take longer (four to nine months). In one case the diffuse pigmentation of the face and the pigmentation of the nail-beds and palate are still present although considerably reduced 14 months after "Camoquin" administration had ceased. Another man, who took "Camoquin" as a suppressive regularly each week for two years and then intermittently for approximately three months of each year for the last four years, still had slight pigmentation of the palate and nail-beds.

No subjects with pigmentation resembling that described above were seen among the 150 adults who either were not taking any malarial suppressive drug or were taking chloroquine, proguanil hydrochloride or pyrimethamine. (Two people who had been taking quinacrine hydrochloride for many years have been seen elsewhere with an identical greyish-blue pigmentation of the face or nail-beds.)

TABLE I.

The Incidence of Pigmentation in Relation to the Duration of "Camoquin" Malarial Suppressive Therapy in 100 Adults of Both Sexes.

Duration of Course in Months.	Number of Subjects Examined.	Number Showing Pigmentation at One or More Sites.
1 to 11	36	1
12 to 23	14	6
24 to 35	22	13
36 to 47	10	6
48 to 59	10	8
60 or more	8	6
Total	100	40 (40%)

Children.

Two hundred and ninety-one children were taking "Camoquin" regularly for periods of time which varied from two months to over five years; 12 of these children (six male, six female) showed evidence of pigmentation of the palate similar to that seen in the adults. Its incidence increased with the duration of the course (Table II) and showed no sex difference. No evidence of pigmentation of the face or nail-beds was found in children.

The incidence of pigmentation of the palate among 94 children who had been taking "Camoquin" regularly for three years or more was 11·7%. No subjects with pigmentation of the palate, similar to that described above, were seen in the 423 children examined who either were not taking any malarial suppressive drug or were taking chloroquine, proguanil hydrochloride or pyrimethamine.

DISCUSSION.

The pigmentation described appears to be similar in its distribution, appearance and incidence with that which was described in adults who had been taking quinacrine hydrochloride ("Atebrin") as a malarial suppressive (Barr, 1944; Lippard and Kauer, 1945; Lutterloch and Shallenberger, 1946; Sugar and Waddell, 1946). The pigmentation with quinacrine hydrochloride was noted only in the nail-beds of the fingers and toes, on the hard palate, on the tip of the nose and on the alæ nasæ. However, pigmentation of the cartilages of the epiglottis and trachea was also reported (Sugar and Waddell, 1946). No mention is made in these reports of a diffuse grey facial pigmentation due to suppressive quinacrine hydrochloride therapy, but I have observed a rather extensive greyish-blue pigmentation of the nose and adjoining parts of the face, similar to that seen with "Camoquin", in a man who had been taking quinacrine hydrochloride for many years.

The pigmentation due to quinacrine hydrochloride was noted in troops in the South-West Pacific area and in Europeans in West Africa (Findlay, 1947). It took seven months to develop, had an incidence of 63% in those who

had been taking quinacrine hydrochloride for over eighteen months (Lippard and Kauer, 1945) and usually disappeared four to nine months after the drug was ceased. There is no record of any investigation of its incidence in children. In two cases only, biopsy of the palate was performed, and the pigment found was thought to contain iron and possibly to be hæmosiderin (Lippard and Kauer, 1945). The peculiar distribution and localization of the pigment could not be explained (Lutterloh and Shallenberger, 1946).

The occurrence of pigmentation during malarial suppressive therapy with "Camoquin" has not been noted elsewhere, but three cases of diffuse melanosis of the skin due to "Camoquin" have been reported in patients with chronic lupus erythematosus who were receiving 300 to 400 mg. of "Camoquin" per day (Young, 1958). After three to four months of continuous treatment, they were noted to have a yellowish-brown to greyish-brown discoloration of the skin, particularly marked on the face and on the parts of the body exposed to the light. One subject had pigmentation of the mucous membrane of the anus and vulva. No pigmentation of the mouth was noted. The sclera of these patients was also noted to be yellow.

TABLE II.

The Incidence of Pigmentation of the Palate in Relation to the Duration of "Camoquin" Malarial Suppressive Therapy in 291 Children of Both Sexes.

Duration of Course in Months.	Number of Children Examined.	Number Showing Pigmentation.
1 to 11	81	0
12 to 23	64	1
24 to 35	52	0
36 to 47	45	2
48 to 59	37	6
60 or more	12	3
Total	291	12 (4%)

In the present investigation, two adults have had biopsies performed on them. One biopsy specimen was taken from the face, and the pigment here was thought to be melanin. The second biopsy specimen was taken from the mucous membrane of the palate. The pigment here did not contain iron, but a complete report on this biopsy is not yet to hand. It seems highly probable that the pigment in the mucous membranes will also prove to be melanin.

The fact that both quinacrine hydrochloride and "Camoquin" can produce an almost identical pigmentation in these very unusual sites (nail-beds, palate, nose and alæ nasæ) suggests that the pigmentation has the same cause in both cases. This may be some common breakdown product of their metabolism in the body, or it may be related to the fact that, unlike the other anti-malarial drugs in current use, both these compounds are yellowish dyes. Why the pigmentation should occur in such unusual sites is not known; nor is the lack of pigmentation of the face and nail-beds in children explained; but answers to these questions may provide information of fundamental importance to a better understanding of pigmentary metabolism.

Except when the pigmentation occurs on the face or is particularly dense in the nail-beds, most men are unconcerned about it and are often unaware of its presence. Women, however, are very conscious of its bad cosmetic effects, particularly when the face or the nail-beds of the fingers are involved. Its occurrence on the face in both sexes has given rise to fears of general disease and may lead to expensive medical investigations and admission to hospital to exclude such diseases as hæmochromatosis or Addison's disease.

If the use of "Camoquin" as a malarial suppressive is contemplated in adults for a period of over two years, the possibility of pigmentation complicating such therapy has to be kept in mind. "Camoquin" now must always be considered as a possible cause in the differential diagnosis of a diffuse grey facial pigmentation, and inquiry about previous and continuous administration of this drug should always be made in such cases.

SUMMARY.

The appearance and incidence of a greyish pigmentation of the nail-beds, palate and face occurring in 100 adults and 291 children of both sexes, taking "Camoquin" regularly as a malarial suppressive, are described.

The similarity of this pigmentation to that which occurred during malarial suppressive therapy with quinacrine hydrochloride is noted and commented on.

ACKNOWLEDGEMENTS.

I wish to thank Dr. F. D. Schofield, Assistant Director (Medical Research) of the Department of Health, and Dr. R. H. Black, of the School of Public Health and Tropical Medicine, Sydney, for their criticisms and suggestions in the preparation of this paper; Dr. W. Symes and Mr. P. Barnes, of the Department of Health, for the coloured photographs; and the Director of the Department of Public Health, Territory of Papua and New Guinea, for permission to publish this paper.

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ADDENDUM.

Dr. D. Torpy, of the School of Public Health and Tropical Medicine, Sydney, has supplied the following report on a biopsy from the pigmented palate of a man who had been taking "Camoquin" for three years:

The material was formalin fixed and sections were prepared in 5 and 8μ thicknesses. The material was over-fixed. For this reason it was not possible to obtain a result with oxidative enzymes (e.g., dopa oxidase).

The staining results are as follows:

1. Eosin-hæmatoxylin: Pigment material scattered. Marked accumulation in the sub-epithelial zone where it was present both intra- and extra-cellular. Colour: v. dark brown to black.
2. Decolorization:
   H_2O_2 15% for 8 hours: No effect.
   H_2O_2 15% for 24 hours: Decolourized.
3. Sudan Black B: Negative.
4. Schmorl: Positive.
5. Nile Blue: Dark Green.
6. Fontana: Positive (darkening).
7. Benzidine: Negative.
8. Perl: Negative.
9. Gmelin: Negative.
10. Lillie: Positive.

From these results it appears that the pigment present is a melanin.

LEGENDS TO ILLUSTRATIONS.

FIGURE II.—Dark-grey pigmentation of the palate in a child, aged eight years, who had been taking "Camoquin" for over six years.

FIGURE III.—Showing dark-grey pigmentation of the alæ nasæ in a man who had been taking "Camoquin" for three and a half years.

FIGURE IV.—Diffuse grey facial pigmentation in a man who had been taking "Camoquin" for three and a half years.

THE ROLE OF THE ELECTROCARDIOGRAPH IN ISCHÆMIC HEART DISEASE.

By J. J. HURLEY, M.B., B.S., AND H. B. KAY, M.D., M.R.C.P., F.R.A.C.P.,
Melbourne.

ISCHÆMIC HEART DISEASE is the direct result of atherosclerotic changes in the coronary arteries which lead to narrowing and in many cases, with or without thrombosis, to complete occlusion of these vessels. The myocardium, as a result of these pathological changes, may show infarction of varying extent, limited or massive and transmural, focal areas of necrosis, fibrosis either localized or diffuse, or no discoverable abnormality at all. The presence of these pathological changes or of the purely functional state of ischæmia modifies the normal electrical activity of the heart muscle and may be reflected in the electrocardiogram. Unfortunately, many abnormal patterns thus observed are not specific for ischæmic heart disease. Diagnosis must be based therefore on a careful consideration of the patient's history and not upon an electrocardiographic abnormality, except, of course, the classical pattern of acute myocardial infarction, which for practical purposes is pathognomonic of that condition.

Pain, the cardinal symptom, may depart from the classical description in character, in distribution and in the factors that excite and relieve it. Therefore the physician who is consulted by a patient with chest pain and is in reasonable doubt as to its nature turns to the electrocardiogram for assistance. To what extent his expectations may be realized is the subject of this article.

Our conclusions are based upon study of the conventional twelve-lead electrocardiogram. At present this technique has, in our opinion, the most detailed correlation of abnormal findings with autopsy data. It is essential that the electrocardiograph be properly used, for faulty technique in lead application or defects in the machine itself may produce abnormalities which may be confused with those found in organic heart disease (Figure I).

ELECTROCARDIOGRAPHIC CHANGES IN CORONARY ARTERY DISEASE.

The electrocardiographic changes associated with coronary artery disease may be discussed under three headings: (i) recent infarction; (ii) old infarction; (iii) changes believed to be associated with myocardial ischæmia.

Recent Infarction.

The classical change in recent infarction is an abnormal pattern involving the *QRS* complex, the *S-T* segment and the *T* wave (Figure IIa).

Abnormal *Q* waves appear in leads facing the area of infarction. They are both deeper and wider than the normal *Q* wave, and are often slurred or notched (Goldberger, 1953). The *S-T* segment in leads facing injured muscle becomes elevated, and its contour is usually, but not invariably, abnormal. There is commonly reciprocal depression in leads facing the surface of uninjured muscle.

After a period of time, often varying widely from case to case, the elevated *S-T* segment returns to the base line, and the corresponding *T* wave inverts and evolves in characteristic fashion. The degree of inversion waxes until a stage of maximum negativity is reached. Thereafter the abnormal *T* wave wanes, at least to some degree, and in the stabilized graph a variable degree of inversion may persist or the *T* wave may be normal. Such an evolution of the *S-T* segment and *T* wave in the presence of abnormal *Q* waves provides indisputable evidence of recent infarction, and can be studied adequately only in serial tracings.

Normal evolution may be modified by extension of the infarct, or by the presence of old infarction or of complications—for example, pulmonary embolism or arrhythmias. On occasions, too, it may be delayed for reasons that are not quite clear, the *S–T* segment remaining elevated for weeks before it returns to the base line and the corresponding *T* wave begins to invert.

The *S–T* segment may have been returned to the base line when the first electrocardiogram is taken. Evidence of recent infarction will then be provided by the progressive evolution of the *T* wave in conjunction with abnormal *Q* waves.

This pattern of recent infarction is one of the most positive findings in clinical medicine—a point of some importance when it is remembered that an infarct may be clinically silent. However, a normal electrocardiogram does not exclude infarction, recent or otherwise. It may be normal at the onset of clinical symptoms of infarction and remain so for days before positive electrocardiographic evidence appears. Also the electrocardiogram may remain normal, or atypical, if the infarct is small, or if the exploring electrode does not "face" the infarcted area of heart muscle — as, for example, in high lateral infarction, or in posterior infarction when the heart is in the electrically horizontal position (Myers, 1949).

Elevation of the *S–T* segment often of abnormal contour points strongly to recent infarction (Figure IIa). In a single record, however, the evidence is not conclusive.

A variable degree of *S–T* segment elevation may be normally present in Leads V_1 and V_2. This may be exaggerated and the segment may assume an abnormal contour if the chest lead is improperly applied (Figure I). In these leads, elevation also is often marked in association with left ventricular preponderance and left bundle branch block patterns (Figure II, *b* and *c*), and though the degree of elevation may vary widely from time to time, evolution of the pattern characteristic of infarction does not occur. Digitalis may likewise cause *S–T* segment elevation, often of domed contour, in leads exhibiting an *rS* or *QS* complex. Finally, the segment may remain permanently elevated when a ventricular aneurysm develops, and it may without this development remain elevated for months or years for reasons that are not clearly understood.

S–T segment elevation occurs in acute pericarditis (Figure II*d*). It is often slight in degree, on occasions barely discernible, and recognized in retrospect only when serial tracings are studied. The contour of the elevated segment is frequently quite straight, or shows at most a slight upward concavity. Reciprocal *S–T* segment depression does not occur. Abnormal *Q* waves are not associated with the *S–T* segment changes unless there is coincidental infarction. When the elevated *S–T* segment returns to the base line, the corresponding *T* wave may invert, as in infarction, but seldom deeply, that is, to a degree greater than 5 mm. (Figure III*d*). Frequently these findings are most marked in Lead aVL, in standard Lead I and in leads V_5 and V_6. Although the evolution of the two patterns is similar, in acute pericarditis it is frequently of shorter duration and, in general, less spectacular than in the ischæmic patterns of coronary heart disease. Whilst there are features peculiar to pericarditis—for example the presence of *S–T* segment elevation in both unipolar limb Leads aVL and aVF, and in all the standard and precordial leads, they are by no means always found, and there will be occasions when electrocardiographic differentiation between the two patterns will be impossible

and diagnosis must be established on purely clinical grounds.

T Wave Changes Only.

Although the abnormal *Q* wave is diagnostic of infarction, if such uncommon conditions as periarteritis nodosa, scleroderma or amyloidosis are excluded, autopsy studies make it clear that infarction can occur without the development of abnormal *Q* waves. However, the diagnosis of recent myocardial infarction cannot be made electrocardiographically on *T* wave changes alone. If an abnormal *T* wave can be accepted as a "coronary" wave, it can be taken to indicate only ischæmia, though infarction may be inferred from the clinical picture and autopsy may confirm its presence. This coronary *T* wave (Figure III*a*), which is found in association with the *QRS* complex and

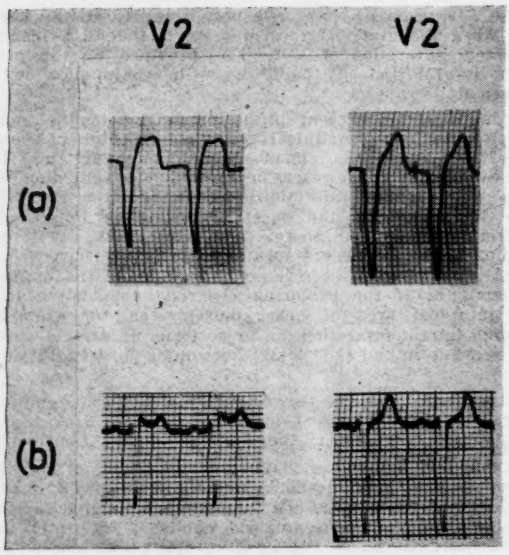

FIGURE I.

Left bundle branch block (Lead V_2) and normal Lead V_2 tracing before and after thorough application of jelly.

S–T segment changes of classical infarction, commonly shows an abnormal symmetry; this, however, is seldom quite absolute. The associated *S–T* segment, except in the stage of injury, is usually isoelectric and often horizontal. It may show an upward convexity, followed by a *T* wave that slopes gently downwards and then rises abruptly to the base line, the so-called cove plane *T* wave. These abnormal *T* waves (the symmetrical and cove plane waves) point to ischæmia but are not pathognomonic of it. Thus the cove plane or coronary *T* wave is commonly found in the transitional zone leads of the left ventricular strain graph, and symmetrical *T* waves may be found in left and right ventricular strain or in pericarditis, to mention only common examples. Nor is ischæmia excluded because the *T* wave does not conform to the classical pattern. Especially is this so in lateral infarction, in which *S–T* segment depression commonly distorts the pure symmetry of the *T* wave. Flat or diphasic *T* waves, waves displaying a terminal dip, *T* waves that are notched or have blunted apices or display a notch on the descending limb may carry the same connotation as the inverted and symmetrical coronary *T* wave, into which, indeed, they may not uncommonly be seen to develop when serial tracings are studied. The significance of an abnormal *T* wave is thus often uncertain.

Especially is this so in Lead aVF and in standard Leads II and III, in hearts with both horizontal and vertical axes. Indeed, at present there is no means of assessing the significance of minor *T* wave changes, with or without *S–T* segment depression in these leads. They may represent ischæmia or early left ventricular strain (in vertical hearts), or may be merely a variant of the normal. However, there is one pattern pointing strongly to the presence of myocardial ischæmia and possibly limited infarction, often subendocardial — namely, the presence of deeply inverted *T* waves of coronary contour in some or all of the anterior chest leads, greater in amplitude than 5 mm., often maximal in Lead V_3, and less deeply inverted or upright in Lead V_6 (Figure VI*a*). Studied in serial tracings, these distinctive, often broad and alarming waves evolve in a characteristic fashion, and after a greater or lesser period of time may return to normal.

The abnormal *T* wave, then, without *QRS* complex or *S–T* segment change, may be a significant finding pointing to the presence of ischæmic heart disease. It is, however, a non-specific change, and when the history and findings are atypical a wide clinical search should be made to elucidate its significance. *U*-wave changes may occur in association with ischæmic heart disease (Evans, 1954). The *U* wave may be inverted and the *T–U* segment depressed; but these findings are rarely of diagnostic value and will not be discussed further in this article.

Old Infarction.

The evolution of the electrocardiographic pattern of recent myocardial infarction ceases after some months or even years. Residual abnormalities are much less characteristic than those found in early tracings. The abnormal *Q* wave of recent infarction may disappear, or may regress and be replaced by a wave that is doubtfully abnormal. Often the only residual abnormality is an inverted *T* wave, which may or may not have classical coronary contour. As a diagnosis of myocardial infarction cannot be made on the basis of abnormal *T* waves alone, it will be clear that only the persistence of an abnormal *Q* wave can provide positive evidence of old infarction.

Goldberger (1953) has stated that *Q* or *Q–S* waves may appear in Lead aVF in three situations: (i) in vertical hearts; (ii) with clockwise rotations of the heart's electrical axis; (iii) in posterior infarction.

The narrow *Q* wave found normally in vertical hearts seldom causes difficulty, because the normal *q–R* ratio is preserved. When clockwise rotation occurs, a *QR* complex is also found in Lead aVR. Such rotation is uncommon in posterior infarction, and an *r–S*, *rSr'* or *Q–S* complex is almost invariably found in Lead aVR unless coincidental anterior infarction is also present. Whatever arbitrary limits are laid down for the width of an abnormal *Q* wave in Lead aVF and Lead III and its depth taken in relation to the corresponding *R* wave, so many *Q* waves remain borderline or equivocal that it is often impossible to assess their significance, and an electrocardiographic report detailing these findings must necessarily be noncommittal.

Q Waves in Lead aVL.

When the lateral region of the heart is infarcted, abnormal *Q* waves may appear in Lead aVL. However, a *QR* complex in this lead, often associated with an inverted *T* wave of coronary contour, is a common finding in the normal heart, and it may vary from tracing to tracing. The diagnosis of antero-lateral infarction therefore should be based primarily upon abnormal findings in Lead I and in Leads V_5 and V_6 taken in the conventional position or in the third left intercostal space.

Q Waves in Precordial Leads.

Abnormal *Q* (*Q–S*) waves appear in precordial leads in anterior infarction. With time they may regress and disappear entirely, or may be replaced by doubtfully abnormal waves which, as in old posterior infarction, create insuperable difficulties in diagnosis. Especially is this so when the *S–T* segment and *T* wave have returned to normal. Confusion also arises from the fact that a *Q–S* complex in Leads V_1 and V_2, often with a greater or lesser degree of *S–T* segment elevation, may be found normally or in electrocardiograms associated with

left bundle branch block (Figure IIc), in the Wolff-Parkinson-White syndrome or in left ventricular preponderance (Figure IIb). The demonstration of an initial *r* wave in leads to the right—for example, V_2R—usually but not invariably indicates that the *Q–S* complex is abnormal. When a *Q–S* complex in Leads V_1 and V_2 is a normal finding, an initial *r* wave may be found in these leads taken at the level of the ensiform cartilage

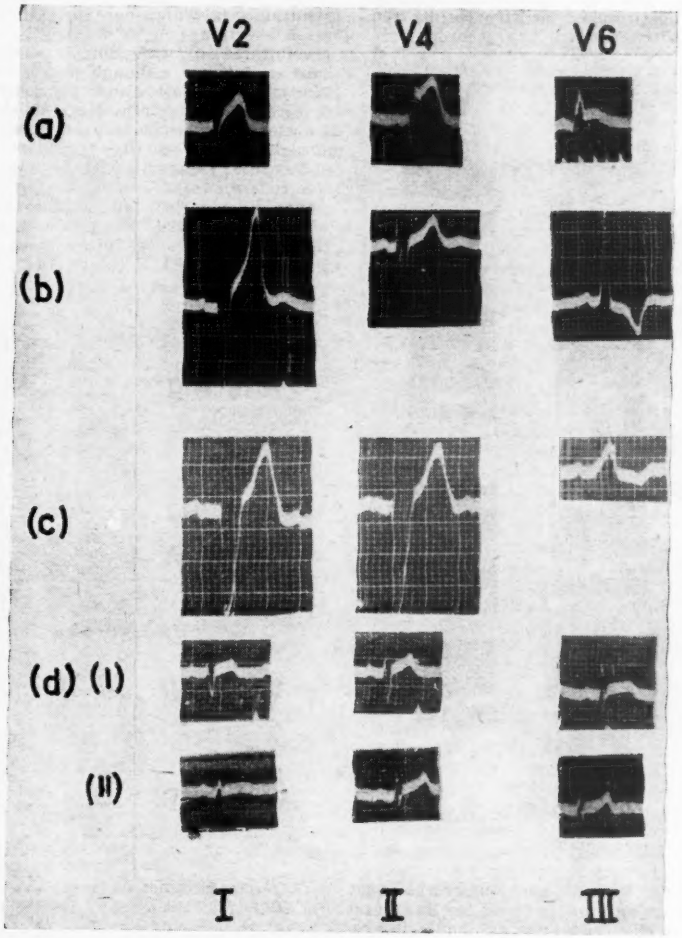

FIGURE II.

S-T segment elevation: (*a*) myocardial infarction; (*b*) left ventricular preponderance; (*c*) bundle branch block; (*d*) I, II, acute pericarditis.

(Surawicz, 1955). The *Q–S* complex of recent infarction may in the course of time be replaced by a triphasic *QRS* complex with a small *q* preceding the *r–S*. According to Myers (1948), this complex in Leads V_1 and V_2 is always abnormal. The broader and deeper the *Q* (*Q–S*, *QR*) waves are and the further they extend to the left, the more likely are they to be abnormal. In lateral infarction, the *Q* wave is uncommonly very deep, and should be regarded as abnormal if it is broad and slurred though the normal *Q–R* ratio is preserved. Normally the *R–S* value increases as the electrode is moved to the left across the precordium. In emphysema, however, with right ventricular dilatation or hypertrophy the *r* wave may become smaller and be replaced by a *Q–S* complex in Lead V_6 without indicating infarction.

The difficulties of diagnosis, especially of old antero-septal infarction, may be better appreciated when it is realized that there is often no unanimity of opinion among experts as to whether a given complex in Leads V_1 and V_2 is a *Q–S* complex and thus consistent with infarction, or an *r–S* complex indicating the normal. It must be emphasized again that *Q* waves, unless clearly abnormal according to conventional criteria, cannot be accepted as evidence of infarction unless they are accompanied by the classical changes in the *S–T* segment and *T* wave.

Myocardial Ischæmia.

Under this heading will be discussed two well-recognized clinical syndromes, angina of effort and acute coronary insufficiency.

Angina of Effort.

In this common syndrome the electrocardiogram is often normal, probably in one-third of cases, and it may remain normal over periods of months or years, even after exercise which precipitates cardiac pain. In a further one-third of cases the tracing is abnormal, but not diagnostic of ischæmic heart disease; for example, atrio-ventricular block, bundle branch block, right or left ventricular strain patterns or non-specific *T* wave changes are seen. In the remainder, the electrocardiogram shows an infarct pattern, usually old, but occasionally recent, the distinctive coronary *T*-wave pattern or *S–T*

segment depression and/or *T*-wave changes highly suggestive of acute coronary insufficiency.

Since the resting electrocardiogram is so often normal, an attempt may be made to induce changes by an exercise test. This is unsatisfactory for many reasons. There is no unanimity of opinion as to the form the exercise should take. The exercise electrocardiogram may remain normal in the presence of ischæmic heart disease even if pain is provoked by the exercise, and there is a wide difference of opinion as to what constitutes a positive result. Consequently, many false-positive results are recorded.

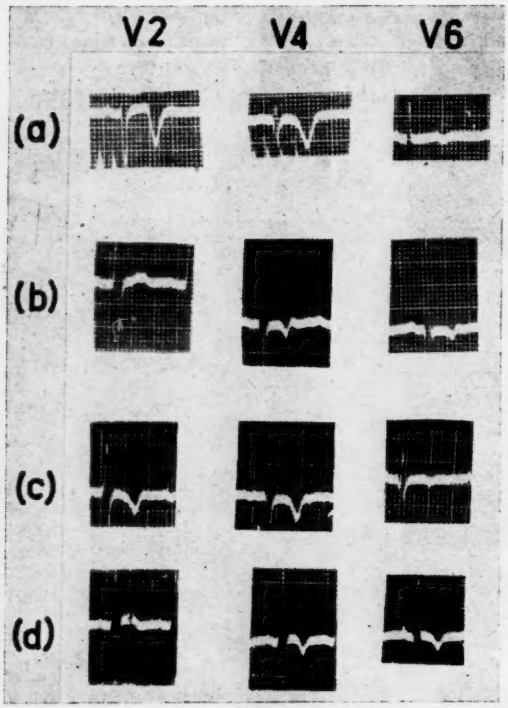

FIGURE III.

T-wave inversions: (*a*) acute myocardial infarction; (*b*) acute rheumatic fever; (*c*) constrictive pericarditis; (*d*) acute pericarditis.

In our opinion no patient with a history of recent onset of angina should be given an exercise test, as infarction may be thereby precipitated. Indeed, recent acute infarction may be marked by no more catastrophic symptoms than the onset of angina of effort. The pre-exercise electrocardiogram should be normal and the patient should not be receiving digitalis, which may produce a false positive result.

In the electrocardiogram taken after exercise (Figure IV), depression of the *S–T* segment of 1 mm. or more in Leads aVL or aVF, or in both, in the precordial leads, most marked in Leads V_4, V_5 or V_6, and in some or all of the standard leads, in association with *S–T* segment elevation in Lead aVR, and with or without concomitant *T*-wave change, in our opinion indicates the presence of anoxia, precipitated by exertion and presumably due to atherosclerosis of the coronary vessels. *S–T* segment depression of lesser degree, 0·5 mm. or more, especially when associated with a change in contour of the displaced *S–T* segment, may, of course, be significant. However, so many borderline or equivocal exercise electrocardiograms are seen that it is better to adhere to fairly rigid criteria before pronouncing the finding positive. After the cessation of exercise the electrocardiogram returns to normal, usually within a period of five to ten minutes.

It must be remembered that spurious depression of the *S–T* segment may occur, especially in Leads aVF, 2 and 3, usually the result of tachycardia and the development of a prominent auricular *T* wave. In such an event, the *P–Q* segment is depressed and the deviated *S–T* segment retains its normal contour.

Acute Coronary Insufficiency.

This term is applied to a sufficiently clear-cut clinical syndrome, in which cardiac pain occurs both at rest and after exertion and is frequently protracted. It may be precipitated by infection, arrhythmias, hæmorrhage or cardiac failure, although more often its onset is spontaneous, enigmatic and unpredictable. The associated features of acute infarction, shock, fever, leucocytosis or an elevated serum transaminase level are minimal or absent. It is usually considered a reflection of subendocardial ischæmia with or without focal necrosis. The electrocardiogram accompanying this clinical state is commonly identical with that found in cases of angina in which the exercise test produces a positive result, except that the changes are often more marked and more protracted (Figure IV, Figure V*a*). It is referred to as the

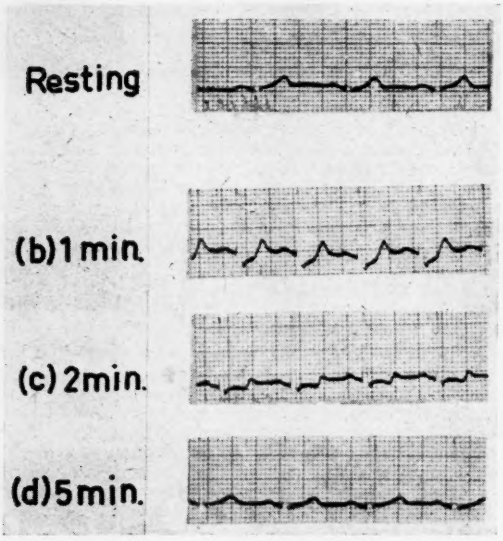

FIGURE IV.

Exercise electrocardiogram: (*a*) resting; (*b*) one minute after exercise; (*c*) two minutes after; (*d*) five minutes after.

anoxic or acute coronary insufficiency pattern. The *S–T* segment depression, which may be extreme, is often associated with *T*-wave changes, usually not gross, or these may occur independently.

The electrocardiographic pattern of acute coronary insufficiency is not unequivocal evidence of significant coronary artery disease. It may occur in shock, in acute hæmorrhage, in pulmonary embolism, or in any clinical state in which a precipitous fall in blood pressure or cardiac output occurs. It is thus non-specific, and should be interpreted only with full knowledge of the clinical situation.

In general, it may be stated that the electrocardiographic findings associated with the clinical state of acute coronary insufficiency when due to ischæmic heart disease are unstable, in sharp contrast to the orderly and progressive evolution seen with uncomplicated recent infarction. *S–T* segment depression of changing degree, altering contours of the deviated segment, and variable *T*-wave abnormalities may persist for hours, days or months. The electrocardiogram may return to normal and then

regress, or at any time, and often without any remarkable change in the patient's clinical condition, the classical pattern of recent infarction may appear. Even more than in angina, it may be difficult to be certain that the onset of this clinical syndrome is not associated with acute infarction. Certainly it may progress to infarction. It therefore demands close electrocardiographic study with serial tracings, along with repeated estimations of the erythrocyte sedimentation rate and the serum transaminase level.

S–T Segment Depression.

S–T segment depression, an important finding in the electrocardiogram of ischæmic heart disease, should be measured with reference to the following *T–P* segment, because the segment may spuriously be depressed by the atrial *T* wave, especially in the presence of tachycardia.

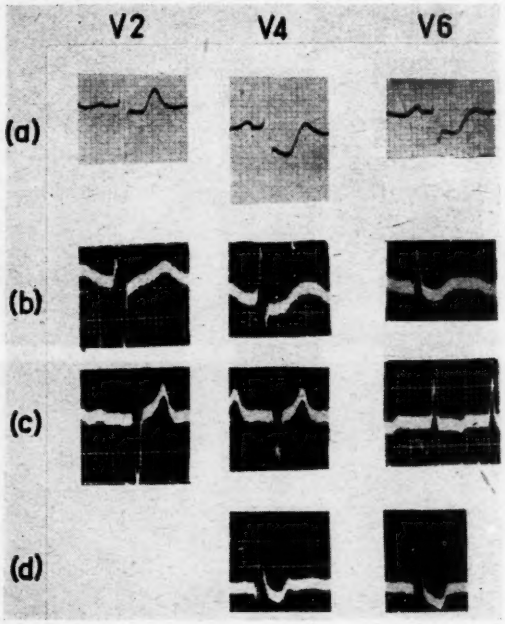

FIGURE V.

S-T segment depression: (*a*) acute coronary insufficiency; (*b*) hypokalæmia; (*c*) left ventricular strain; (*d*) digitalis effect.

Several *QRS* complexes on a perfectly horizontal line should be available for study. When the *S–T* segment is significantly depressed — that is, 1 mm. or more — it is usually associated with an abnormal *S–T* segment contour (Evans, 1954). *S–T* segment depression is most commonly found in association with states of acute coronary insufficiency (Figure V*a*). Cardiographically it has to be distinguished from the common digitalis effect, from hypokalæmia and from left ventricular strain.

Digitalis.—This drug is to the electrocardiogram what syphilis was to clinical medicine—the great imitator. Accordingly it will often be impossible to pronounce abnormal an electrocardiogram showing non-specific *S–T* segment depression, when the disturbing influence of digitalis cannot be adequately assessed. In these circumstances, if it is decided to suspend digitalis therapy, it must be remembered that some preparations—for example, digitoxin—are slowly eliminated, and a period of up to three weeks must be allowed to elapse before it can be decided that the abnormalities are not due to the drug. It is clear that a statement indicating digitalis dosage should accompany every request for an electrocardiogram. Digitalis first depresses the *S–T* segment near its junction with the *T* wave, giving it a downward bowing (Figure V*d*). This downward bowing, in association with the shortening of the *Q–T* interval which digitalis usually produces, provides a pattern that is often characteristic and should not be confused with the coronary insufficiency pattern in which the *Q–T* interval is more usually prolonged. This digitalis effect is associated with an upright *T* wave. As the action of the drug becomes more manifest, the *T* wave becomes at first diphasic and finally inverted.

All distinction between the *S-T* segment and *T* wave may become lost and the pattern becomes less specific. The position is further complicated by the fact that many patients whose electrocardiograms show ischæmic or left ventricular strain patterns are receiving digitalis. A short *Q-T* interval or a *Q-T* interval shortening in serial tracings points to digitalis; but often no more can be said than that a given non-specific abnormality is present and consistent with the effect of digitalis and/or ischæmia, coronary insufficiency or left ventricular strain.

Hypokalæmia.—Disturbances of electrolyte balance, and in particular hypokalæmia, produce electrocardiographic abnormalities often confused with the non-specific abnormalities of ischæmic heart disease. Thus a low serum potassium level may depress the *S-T* segment and invert the *T* wave. However, it produces a significant modification of the *U* wave, notably an increase in voltage and fusion to a greater or lesser degree with the *T* wave, which changes give to the electrocardiogram its peculiar and distinctive character (Figure V*b*).

The electrocardiogram in hypokalæmia may vary widely in respect to the relative changes in the *S-T* segment, the *T* wave and the *U* wave. *S-T* segment depression, usually maximal in left ventricular leads, is not constantly present. The *T* wave, while commonly inverted, may remain normal in all leads. It may remain upright in Leads V_3 and V_4 when it is inverted in Leads V_5 and V_6. A common and highly significant finding is the fusion, more or less complete, of an upright *T* wave with a *U* wave of high voltage. The summit of the fused wave is often broad and, as it were, smudged, and if the union is complete, the illusion is created of a greatly prolonged *Q-T* interval (Figure VIIc). Actually the *Q-T* interval is not prolonged in hypokalæmia except in the presence of associated hypocalcæmia (Lepeschkin and Surawicz, 1952).

With increasing degrees of hypokalæmia the *T* wave may become diphasic or inverted. *Pari passu* with this change, the *U* wave increases in voltage. The depressed *S-T* segment, the inverted or diphasic *T* wave and the augmented *U* wave then take on an appearance that is highly characteristic, the contours being curiously smooth or suave and resembling the letter *S* turned on its side (Figure V*b*).

Superficially, the resemblance to the acute coronary insufficiency pattern is close. When the *T* and *U* waves are of opposite polarity, the *U* wave may masquerade as a *T* wave and the *Q-T* interval be considered to be prolonged. Therefore a careful search should be made of all leads, with special reference to Leads V_3 and V_4, in which the *U* wave is usually of maximum voltage, and to Lead aVL, in which the *U* wave is normally of low voltage, to establish the separate identity of the *T* and *U* waves. The merging of these waves in hypokalæmia is not always complete, and a notch in the equivocal wave may be detected or its apex may be curiously blunted (Figure VIIc).

In many cases, however, especially when tachycardia is present, the *U* wave cannot be clearly identified, and it may be impossible to differentiate between the acute coronary insufficiency pattern and hypokalæmia. The serum potassium level will then usually decide the issue.

Left Ventricular Strain.—This refers to an abnormal electrocardiogram associated with hypertension, aortic stenosis and other conditions imposing an increased burden on the left ventricle. The highly developed pattern seen in association with increased *QRS* voltage in left ventricular preponderance electrocardiograms and indicating left ventricular hypertrophy is frequently highly characteristic, the *S-T* segment being depressed with an upward bowing and the inverted *T* wave strikingly

asymmetrical (Figure II*b*, V_6). These findings appear in leads representing predominantly left ventricular potentials —that is, Lead V_4, V_5 or V_6, and if marked clockwise rotation is present, in Lead V_7 or V_8. In the electrocardiogram showing early left ventricular strain, however, a wide variety of patterns may be seen, differing in respect of the degree of *S-T* segment depression and *T*-wave inversion and of the contour of the displaced *S-T* segment, and the changes may not be constant from record to record. It is frequently unwise to attempt the differentiation of these non-specific changes, some of which may be so slight as to be within normal limits, and it is clearly a crude simplification of the problem to attribute an abnormality to strain in the presence of hypertension and to ischæmia, when a history of chest pain, often equivocal, is given.

qR or *R* complex in Lead aVR and *R-S* or *r-S* complexes in precordial leads, extending to the left as far as Lead V_5 or V_6. However, the pattern is non-specific, and as clockwise rotation is frequently present with antero-septal ischæmia, differentiation of the two electrocardiographic patterns may be impossible.

Recognition of the right ventricular strain pattern is important, because it may appear in association with pulmonary embolism (Figure VIc), spontaneous pneumothorax or massive pulmonary collapse, conditions which may present with many of the clinical features of recent myocardial infarction. Cardiac failure precipitated by vitamin B_1 deficiency may be accompanied by the electrocardiographic pattern of right ventricular strain (Figure VI*b*).

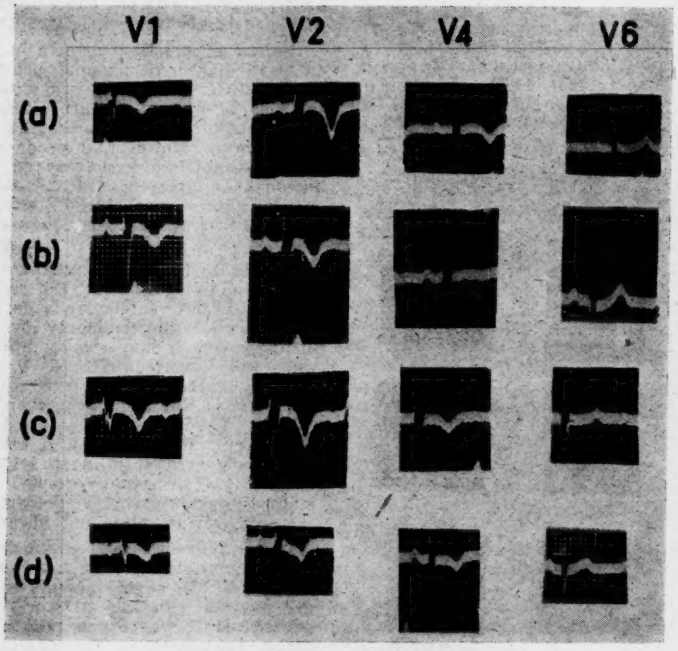

FIGURE VI.

T-wave inversions: (*a*) antero-septal ischæmia; (*b*) beriberi; (*c*) pulmonary embolism; (*d*) right ventricular strain.

In the electrocardiogram of left ventricular preponderance, a *Q-S* complex often with considerable *S-T* segment elevation may be found in Leads V_1 and V_2 and cove plane or symmetrical coronary *T* waves may appear in the transitional zone leads. These findings are not a reflection of ischæmic heart disease.

Right Ventricular Strain.—In right ventricular strain, inverted *T* waves appear in Leads V_2 and V_3, as well as in Lead V_1, in which lead the *T* wave may be normally inverted. In early strain such inverted *T* waves are commonly shallow and show a marked respiratory variation. If the strain is more intense, especially if clockwise rotation is marked, the *T* waves in Leads V_4 and V_5 and even in Lead V_6 may become negative. The *T* waves then are usually more deeply inverted and may be of coronary contour (Figure VI*d*).

However, the spectacular inversion appearing in many cases of antero-septal ischæmia is rarely seen.

The following findings may be present in association with the right ventricular strain pattern: a vertical heart, a deep *S* wave in Lead I, a *P* pulmonale or *P* mitrale in Lead aVF and in standard Leads II and III, and transient right bundle block patterns, partial or complete. Clockwise rotation, often marked, is common—that is, a *QR*,

Finally, it is well to remember that this pattern may indicate no more drastic an abnormality than a depressed sternum or funnel chest.

Bundle Branch Block Patterns.

The classical pattern of infarction will be modified when it is superimposed on an already abnormal pattern—for example, bundle branch block or the Wolff-Parkinson-White syndrome. With preexistent left bundle branch block or the Wolff-Parkinson-White syndrome, abnormal *Q* waves rarely develop. In these patterns also the *S-T* segment is usually depressed and the *T* wave inverted in predominantly left ventricular leads. Thus the classical evolution of the elevated *S-T* segment and inverted *T* wave is obscured and rarely diagnostic. The most important criteria of recent infarction, *Q* waves and *S-T* segment elevation, are therefore lacking, and a diagnosis of infarction can rarely be made electrocardiographically.

Part of the normal left bundle branch block pattern is a *Q-S* complex in Leads V_1 and V_2, often with considerable *S-T* segment elevation and a *Q-R* complex in the transitional zone area. These findings do not indicate infarction. In this pattern (and in the Wolff-Parkinson-White syndrome) the *S-T* segment and *T*-wave components of the electro-

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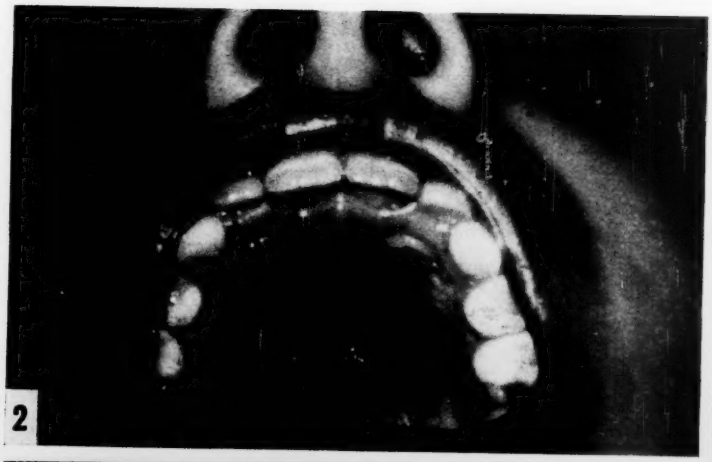

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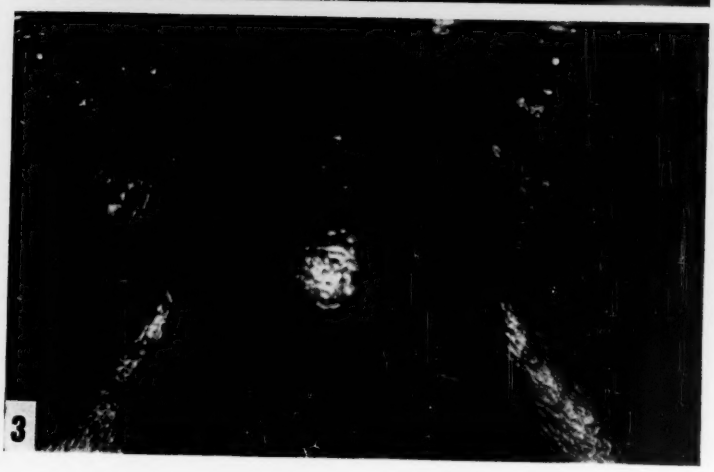

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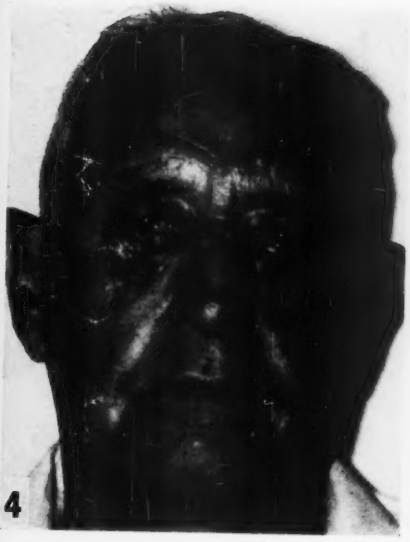

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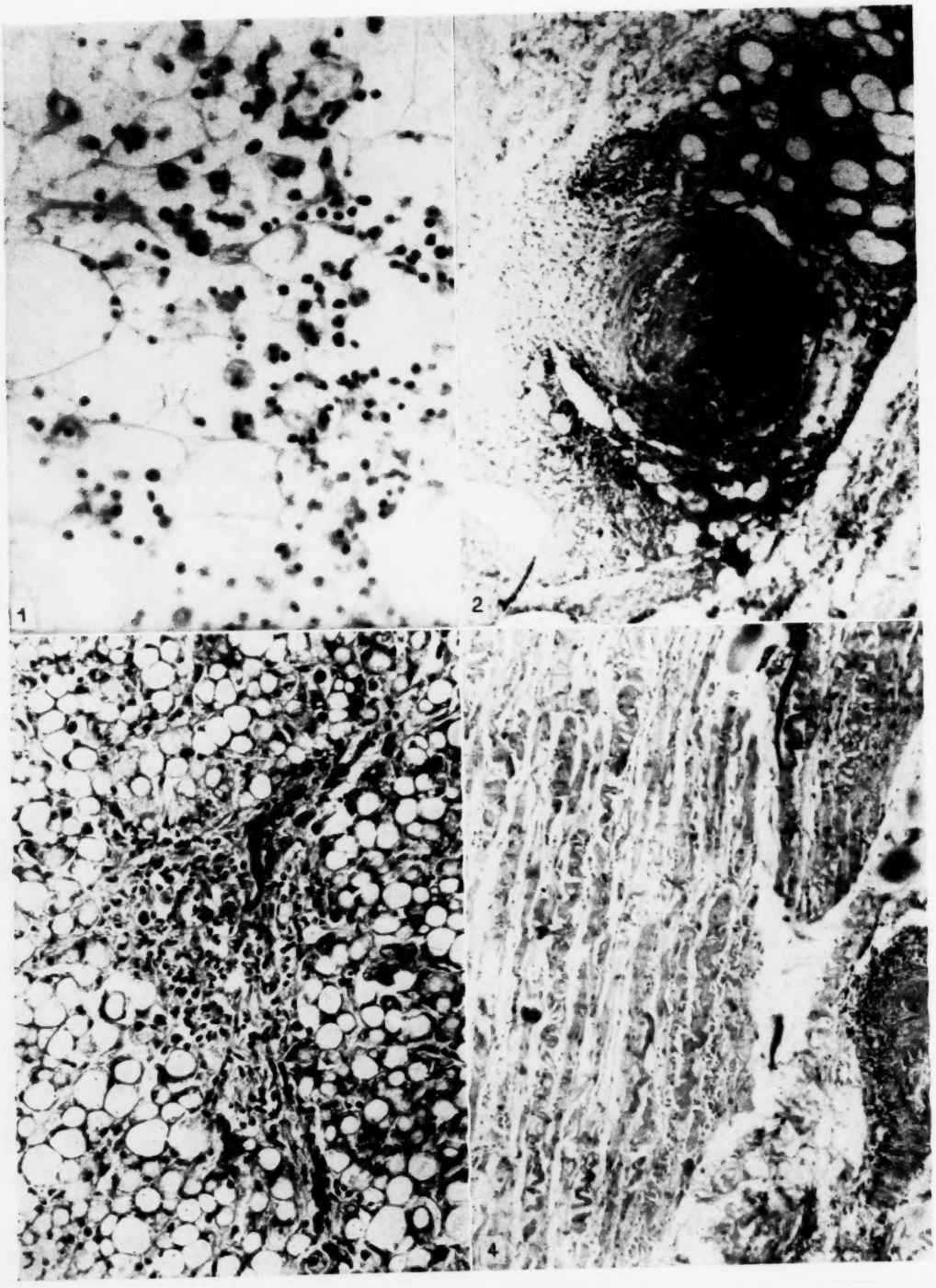
ILLUSTRATIONS TO THE ARTICLE BY P. J. KIERNAN AND H. G. BURGER.



cardiogram are often unstable. However, these changes are erratic and show no evolutionary trend. They cannot be accepted as evidence of recent infarction. It is thus fortunate that acute infarction seldom presents with a left bundle branch block pattern.

When right bundle branch block is present and infarction occurs, either anterior or posterior, the electrocardiogram usually shows the pattern of both right bundle branch block and infarction; that is to say, significant *Q* waves develop and the characteristic evolution is observed. A small *Q* wave is often seen in Leads V_1 and V_2 in right bundle branch block without infarction. If *CR* leads are taken, a small initial *r* wave may be registered, indicating clearly that the pattern is that of uncomplicated right bundle branch block.

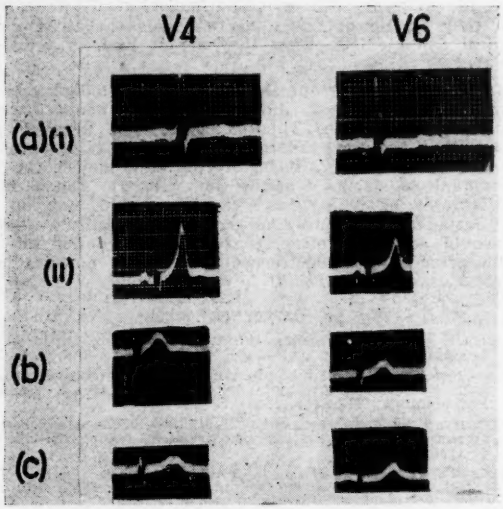


FIGURE VII.

T-wave changes: (*a*) myxœdema, (i) before treatment, (ii) after treatment; (*b*) quinidine effect; (*c*) hypokalæmia.

Traditionally it has been agreed that right bundle branch block as an isolated finding is compatible with normal life expectancy. There is reason to believe that in similar circumstances, left bundle branch block may be equally benign. It is an electrocardiographic abnormality only, and its importance or otherwise must be assessed purely on clinical grounds.

DISCUSSION.

The electrocardiograph, once an eclectic instrument in the hands of the few, has now become standard equipment in many large general practices. We have no quarrel with this; indeed, it is idle to complain about the tyranny of fashion in whatever form it takes. The electrocardiograph is clearly here to stay, and we must make the best of it. That the best is not being made of it is to state the position in the mildest possible terms.

The reasons for this are not hard to find. There is no clear appreciation of the limitations of the electrocardiograph as a diagnostic tool. In hospital practice a request is often received—too often, it may be said—for an electrocardiogram to "exclude" ischæmic heart disease. A normal electrocardiogram does not "exclude" ischæmic heart disease. Neither can it be assumed that ischæmic heart disease is present if the electrocardiogram is abnormal. The basis of abnormal *S-T* segment or *T*-wave changes may be physical, infective, toxic, hormonal or biochemical, as well as ischæmic. These abnormalities appear in electrocardiograms taken for the further study of suspected heart disease, or as a routine procedure as part of a general physical examination when no suspicion of organic heart disease exists. Even when they are clearly studied in serial tracings, their meaning may be far from clear. It is well to repeat, therefore, that an electrocardiographic abnormality, whether it be an arrhythmia, atrio-ventricular block, bundle branch block or non-specific *S-T* segment or *T*-wave change, is not a disease. If the clinical diagnosis of ischæmia is uncertain, the situation is not changed when the electrocardiogram shows non-specific abnormalities.

This should be clear. However, genuine difficulties arise from the fact that many diseases present with clinical features and abnormal non-specific electrocardiograms that closely resemble those found in association with ischæmic heart disease.

For example, symptoms pointing more or less strongly to the presence of myocardial infarction include chest pain, syncope, collapse, arrhythmias of various kinds and congestive heart failure. Pulmonary embolism may present with some or all of these features. It is valid, therefore, to point out, this being primarily a discussion on the electrocardiogram, that non-specific patterns, accompanying a clinical state in which the clinical diagnosis of myocardial infarction is open to the slightest doubt, should prompt full consideration of all conditions entering into the differential diagnosis. These include, in addition to pulmonary embolism, spontaneous pneumothorax, pneumonia, pleurisy, acute pericarditis, dissecting aneurysm and acute upper abdominal emergencies. Acute pericarditis, especially the benign or relapsing form (Figure III*d*), and pulmonary embolism (Figure VI*c*) are of special importance. Clinically they resemble ischæmic heart disease, and cardiographically they show many features in common.

Peripheral circulatory failure commonly accompanies the onset of myocardial infarction, which may be painless. In shocked states generally, therefore, when the cause of shock is not immediately apparent, an electrocardiogram is taken as routine procedure. It frequently shows the pattern of acute coronary insufficiency. Less commonly *T*-wave changes may be found. It has already been emphasized that these patterns are non-specific. Pulmonary embolism, acute hypotensive states, hæmorrhage, abdominal emergencies (especially acute pancreatitis) or operative or post-operative shock may be associated with such abnormal electrocardiograms, which are frequently complicated by arrhythmias.

It is clear, therefore, that acute circulatory failure accompanied by non-specific *S-T* segment or *T*-wave changes should not be attributed to ischæmic heart disease until other causes of such failure have been excluded. Especially is this so in relation to operative or post-operative shock, which is seldom due to myocardial infarction.

Congestive cardiac failure in the absence of valvular disease or hypertension is often assumed, by a process of exclusion, to be due to ischæmic heart disease, and support for this hypothesis may be given by the appearance of abnormal *T* waves in the electrocardiogram. Such assumptions are dangerous. Infection, beriberi, post-tachycardia syndromes and pericarditis (Figure III*c*) may be the operative ætiological factors, and intrinsic myocardial disease may be minimal or absent.

The abnormal *T* wave due to ischæmia may be flat, notched or diphasic, as well as of more typical coronary contour. These waves are non-specific. There is no clear way of differentiating them from the abnormal *T* wave associated with myxœdema or avitaminosis, for example, in a single tracing. Their evolution is, of course, different. The abnormal *T* wave of myxœdema (Figure VII*a*) or avitaminosis will return to normal with appropriate specific treatment.

The clinical state will thus often determine the significance of abnormal findings. *T*-wave inversion, seldom deep, but nevertheless of coronary contour, may occur in association with many acute infections, both bacterial and viral. In rheumatic fever (Figure III*b*), acute nephritis, rheumatoid arthritis, disseminated lupus erythematosus and other collagen diseases, similar changes may be found. Influences modifying the physico-chemical activity of the myocardial cell may produce *S-T* segment or *T*-wave

changes. These influences include drugs, of which digitalis, quinidine (Figure VIIb), "Pronestyl", adrenaline and emetine are the most important, and also hypoglycæmia, hyperventilation, anaphylaxis, sensitivity reactions, alteration of the serum potassium (Figure VIIa) and calcium levels and Addison's disease.

We have recently had the opportunity of studying a remarkable series of *T*-wave changes in the electrocardiograms of patients receiving treatment for amœbiasis with emetine. It is clear, then, that the *T* wave, being sensitive and labile, is responsive to many influences, often trivial. It may become inverted after a carbohydrate-rich meal or through changes in posture.

Finally, it must be remembered that abnormal *T* waves, for which no explanation at all can be given, may be found in the electrocardiograms of normal healthy individuals.

The abnormal *T* wave, then, is certainly not a disease. The "cardiographer" is, however, constantly asked to report upon an electrocardiogram showing no more than a non-specific *T*-wave change. The practice, as widespread as it is unfortunate, of reporting non-specific *S-T* segment and *T*-wave abnormalities as being "consistent with the presence of ischæmic heart disease" does not serve the best interests of clinical medicine.

No physician should lend unwarranted support to what may well be an even more dubious clinical diagnosis. A doctor who refers a patient for an electrocardiogram does so in the hope of receiving information of value. However, a "cardiographer" is seldom in a position to make an oracular pronouncement. Often he can say no more, or indeed should say no more, than that an electrocardiogram is abnormal and that the findings are non-specific. Such non-committal reports may be considered unsatisfactory; but we do not know of any other special investigation that will consistently provide an unequivocal answer to a clinical problem, and certainly the electrocardiograph is no exception. It possesses no magical properties, as some imagine, and it cannot be allowed to usurp the function of the clinician. If it does so, great and irretrievable harm will be done.

It is understood, of course, that clinical judgement, being fallible, may err. That, however, is not our problem. We are chiefly concerned to point out how the electrocardiogram may mislead those not familiar with its potentialities and its limitations. Paradoxically, on the one hand an unwarranted diagnosis of ischæmic heart disease is made on evidence largely supplied by a non-specific inversion of the *T* wave, while on the other it is rejected because the electrocardiogram is normal or shows minor changes in the *S-T* segment and *T* wave that are statistically within normal limits. These minor changes must be reported as normal by the cardiographer who is not in possession of a full and accurate clinical history, and who has no opportunity of following the patient's clinical progress—a situation constantly arising. These minor changes may, however, be significant. A common problem in practice is the management of a patient with a recent history of suggestive but atypical chest pain. Evidence is accumulating that proper management of the patient with rest and anticoagulants may favourably influence the course of his illness. Impending infarction may, in fact, be averted. A report that an electrocardiogram is normal is often considered, quite wrongly, to weigh heavily against a diagnosis of ischæmic heart disease. This misunderstanding is unfortunate. Minor *S-T* segment changes, which may be within normal limits, may, in the light of the patient's progress, acquire significance. When followed in serial tracings, they may evolve into patterns of diagnostic importance. Therefore, patients with a history of recent onset of suspected angina, or presenting with the syndrome of acute coronary insufficiency, should be closely followed with serial electrocardiograms and routine laboratory tests.

SUMMARY.

The position of the electrocardiograph in relation to the positive diagnosis of ischæmic heart disease may be fairly summed up as follows. The pattern of acute infarction and its classical evolution is for all practical purposes pathognomonic. If the *T* wave alone is involved, and if the waves are deeply inverted and of coronary contour and evolve in expected fashion, ischæmia is the most likely diagnosis, and the onus is on the physician to disprove it if the clinical history and findings are equivocal. The electrocardiographic pattern of acute coronary insufficiency may be highly suggestive. However, it cannot always be clearly differentiated from other patterns showing *S-T* segment depression, and if it can be accepted as being due to subendocardial ischæmia, it does not necessarily follow that it is of primarily cardiac origin and not secondary to other states associated with hypotension. Therefore, the most careful clinical observation and judgement are essential for its proper interpretation. All other non-specific *S-T* segment and *T*-wave abnormalities add little or nothing to the final clinical assessment, and they should not be used to decide the issue when clinical doubt exists. That they are so used is a matter for regret.

The electrocardiograph is widely used today, and if current trends are any indication, it will be used more widely in the future. This being so, it is necessary to repeat that the present status of electrocardiography is far from satisfactory. It is, of course, a matter for the individual to decide whether he will use the electrocardiograph or not. However, he should not do so without adequate training, at present not readily available. It would seem, therefore, that some instruction in the basic principles of electrocardiography, at post-graduate level at least, is desirable.

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FATAL PANNICULITIS.

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THE disease "relapsing nodular non-suppurative panniculitis" was so named by Weber in 1925, when he described a case which he considered similar to the cases reported by Pfeiffer (1892) and by Gilchrist and Ketron (1916). Three years later, Christian (1928) added another case to the literature and justified the addition of "febrile" to the title. Brill (1936, referred to the condition as Weber-Christian disease in "Medical Papers Dedicated to Henry Asbury Christian", and this eponym has persisted. Shuman (1951) reviewed 54 cases of the disease, and since then at least a further 12 have been reported. The characteristic features of the disease include the onset of fever with a temperature of 102° F. or higher, together with the appearance of numerous painful subcutaneous nodules predominantly on the extremities, in particular the thighs and the buttocks; but the subcutis anywhere and even the internal fat deposits may be affected (Spain and

Foley, 1944). These nodules of inflamed fat vary in diameter from 1 to 12 cm. and appear episodically. The overlying skin is usually erythematous, but may not be fixed to the underlying lesion. Healing, which may take weeks or months, is characterized by pigmented skin foci with depression due to the disappearance of the subcutaneous fat. Rothman-Makai disease is a similar form of spontaneous panniculitis, which differs from Weber-Christian disease in that the lesions are symmetrical and that there is no involvement of the overlying skin or subsequent atrophy.

Recurrences with similar clinical findings take place at intervals varying from days to years. Occasionally cystic liquefaction with discharge of cloudy fluid occurs, leading to ulceration. Acute abdominal pain with nausea and vomiting may coincide with the appearance of the nodules, and there may also be myalgia and arthralgia.

The age incidence in the reported cases varies from six months in the male patient described by Carol (1941) to 70 years in the woman patient of Sweitzer (1941). However, the peak incidence is in women from the second to the fourth decade, and the case to be described falls into this group.

The disease has no pathognomonic features, the diagnosis depending on the presence of a consistent clinical picture with the appearances of panniculitis on histological examination. These consist mainly of lymphocytic infiltration between the fat cells, with foamy macrophages resulting from phagocytosis of the fat, and the formation of an occasional giant cell. The blood chemistry and the blood have shown no specific changes, leukopenia being frequently noted as well as normal counts and leukocytosis. Liver function tests have been found to give abnormal results in a few cases of the disease (Oram and Cochrane, 1958; Kritzler, 1941).

Autopsy has been carried out in less than a dozen cases to date, in five of which (Miller and Kritzler, 1943; Mostofi and Engleman, 1941; Hanrahan *et alii*, 1951; Steinberg, 1953) the patients' death could be attributed to their disease. The most consistent, although not constant, findings *post mortem*, apart from the skin lesions, have been fatty change in the liver and hydropic degeneration of the adrenal cortex. Necrosis and inflammation in a liver which had undergone an extreme degree of fatty infiltration were a feature of the case to be described.

The occasional involvement of tissues other than the skin was emphasized recently by Oram and Cochrane (1958). Inflammation of the fat in the omentum, epicardium, perinephric tissues and many other regions internally was described by Mostofi and Engleman (1947) and others. Moderate fat accumulation in the liver is not an unusual finding in any febrile disease; but a severe degree of change has been found in several autopsies in Weber-Christian disease, and in Oram and Cochrane's case (1958) massive hepatic enlargement, confirmed at laparotomy as being due to fat, actually preceded the appearance of typical skin lesions. Splenomegaly is an inconstant although frequently reported finding, and muscle pains and regional lymphadenopathy may also occur (Pfeifer, 1892; Ziegert, 1940). Bone-marrow involvement with patchy fibrosis, fat necrosis and lymphocyte infiltration has been reported in one case (Steinberg, 1953) and a destructive bony lesion in another (Delar and Martz, 1958).

The disease in the form restricted to the panniculus does not appear to have a high mortality. Of 27 patients with skin lesions alone, only two died, one of the deaths being due to intercurrent tuberculosis. The "systemic" disease carries a much higher mortality, and is said to be recognizable clinically by severity of symptoms, by pancytopenia or an increase in circulating mononuclear cells, or by a decrease in the prothrombin time with a positive response to the cephalin flocculation test.

The ætiology of the disease is unknown, and a full discussion of the factors concerned may be found in Hallahan and Klein's article (1951). Some cases, including that of Weber (1925), have been attributed to halogen sensitivity, and administration of iodide in Ungar's case (1946) produced an exacerbation of symptoms. No constant bacterial or viral agent has been isolated from the lesions, although Birrell (1952, 1953) reported a case of "Weber-Christian syndrome" due to leprosy. Trauma and avitaminosis have been cited as causes.

Kennedy and Murphy (1949) suggested that the changes in the panniculus were secondary to vascular disturbance. It is true that arteritis and phlebitis have been described in numerous cases of this disease; but in others of equal severity merely a perivascular collection of cells without involvement of the arterial wall has been found, or no change around the vessels whatsoever, and it is thought that other explanations for the disease are more likely. In this context the work of Duran Reynals (1946), who produced in rabbits areas of panniculitis distant from the sites of injection of various bacteria, is relevant, and would suggest some abnormal antibody-antigen reaction. From this aspect it is interesting to note the high incidence of rheumatic fever and drug sensitivity in these patients. Taranta *et alii* (1958) reported a series of three children suffering from rheumatic carditis, who developed nodular panniculitis on ceasing to take prednisone, which had been given in a dose of 40 to 60 mg. per day. The panniculitis responded to reinstitution of steroid therapy. A similar series was reported by Smith and Good (1956), one of their patients, however, being under treatment for leukæmia. It could be postulated that in these cases an antigen-antibody reaction of the kind suggested was unmasked by the withdrawal of steroid therapy. More and Movat (1958), in discussing polyarteritis, regard evidence of proliferation of plasma cells in the hæmatopoietic tissues, and in the actual lesions, as the morphological equivalent of antibody production, and as morphological evidence for hypersensitivity as an ætiological factor in arterial necrotizing lesions. In the present case of panniculitis, plasma-cell accumulations were found in the liver, spleen and muscles, as well as in the skin lesions, in which some arteritis is present. However, the proplasmocytes which they describe were not found.

Report of a Case.

The patient was a housewife, aged 40 years, who had experienced no previous serious illnesses. Four months before her admission to hospital, which was in May, 1957, she observed a small brown area in the skin of the right leg below the knee. It grew larger, thicker and darker, and eight weeks before her admission became ulcerated and discharged some white material. She considered that local applications brought about temporary improvement, but the lesion had again become worse on her admission. During the same four months she noted a few small lumps in the skin of both legs and over the right ribs, tender at first but later painless. Over the eight weeks when the initial lesion was ulcerated, the patient experienced frequent night sweats and morning headaches, the latter being relieved by salicylates. Her referring doctor stated that she had been febrile (temperature up to 101° F.). On systematic interrogation, the only complaints were of recent pruritus vulvæ and mild burning on micturition.

Examination revealed the patient to be a woman of healthy appearance, with a blood pressure of 115/75 mm. of mercury, a pulse rate of 120 per minute and temperature of 98° F. On the inner side of the right leg, below the knee, there was a round ulcer 4 cm. in diameter, with deeply undermined edges, based on the deep fascia and muscle, these being covered by a black leathery slough. There was a second smaller ulcer below the right inguinal ligament, 1·5 cm. in diameter. Scattered over the left leg, and present also on both arms and on the chest wall, there were a number of flat brown lesions, 1 to 2 cm. in diameter, slightly indurated, with a scaly dry surface. The remainder of the examination revealed no abnormality. Two days after her admission, the patient began to exhibit a swinging temperature (up to 104° F.).

A number of laboratory investigations were undertaken. The hæmoglobin value was 11·9 grammes per 100 ml. The leucocytes numbered 4000 per cubic millimetre, and the differential count revealed no abnormality. The

erythrocyte sedimentation rate was 10 mm. per hour (Westergren). X-ray films of the chest and pelvis showed normal appearances. Microscopic examination of the urine revealed no abnormality. The blood Wassermann reaction was negative. Agglutination tests for typhoid, paratyphoid and brucellosis gave negative results. Swab from the ulcer yielded coliform bacilli only. Repeated blood cultures grew no organisms. Mantoux test (1/1000) produced a negative response. Liver function tests carried out 10 days after the patient's admission to hospital gave the following results: serum bilirubin content, 0·2 mg. per 100 ml.; total serum protein content, 6·6 grammes per 100 ml.; albumin 3·4 grammes, globulin 3·2 grammes per 100 ml. Paper chromatography showed some increase in the gamma fraction. No other abnormality was detected. The serum alkaline phosphatase level was 11 King-Armstrong units. The cephaline flocculation test produced a negative result. A biopsy of a plaque on the back was reported on as follows (Figure I[1]):

> There is infiltration of the subcutaneous fat with lymphocytes and fat filled macrophages. There are areas of necrosis present. Fibrous septa are œdematous and walls of small blood vessels are thickened. One vessel is thrombosed. No tuberculoid granulomata or giant cells are seen in the lesion. The dermis contains a number of lymphocytes, mainly around blood vessels. The epidermis appears intact.
>
> Panniculitis. The appearances are consistent with those of Weber-Christian disease.

The pathological report on a biopsy of a plaque on the foot was as follows (Figure II):

> There is infiltration of the subcutaneous fat with lymphocytes and macrophages. No tuberculoid granulomata or giant cells are seen. The walls of the vessels in the fat are thickened. Some show fibrosis while others are necrotic. There is a perivascular inflammatory infiltrate, many lymphocytes and macrophages being seen, together with occasional eosinophil and neutrophil polymorphs. One vessel is occluded by thrombus. The dermis shows small perivascular inflammatory foci. The epidermis is normal. The appearances are those of Weber-Christian disease, but in view of the vascular changes, polyarteritis nodosa cannot be excluded.

The patient was at first treated with crystalline penicillin (1 mega unit initially, then 600,000 units every four hours) and streptomycin (0·5 gramme twice a day) by intramuscular injection without any clinical improvement. These drugs were therefore discontinued after eight days, and treatment with prednisone (30 mg. on the first day, 20 mg. daily thereafter) was commenced on the fifteenth day after her admission to hospital. There was an immediate fall of her temperature to normal, with an increase in her general well-being, but no significant alteration in the cutaneous lesions. Then she remained afebrile, except for four spikes of temperature up to 100·4° F., for three weeks, during which period, however, she developed superficial thrombophlebitis in the left thigh and several new lesions on the abdominal wall and legs. As these progressed, some became ulcerated, while others healed, leaving flat areas of pigmented wrinkled skin.

On the fifty-eighth day after her admission to hospital the patient developed stomatitis, and two days later had a painless episode of hypotension (blood pressure 75/60 mm. of mercury) with tachycardia, shown by an electrocardiogram to be of supraventricular origin. There was no evidence of myocardial infarction. The hæmoglobin value at this stage was 12·2 grammes per 100 ml., the leucocytes numbered 3000 per cubic millimetre and the erythrocyte sedimentation rate was 6 mm. per hour. A differential leucocyte count gave the following results: metamyelocytes 4%, staff forms 37%, segmented neutrophils 10%, lymphocytes 41%, monocytes 3%, eosinophils 5%. At this time also the left pupil was noted to have become larger than the right. Reaction to light and on accommodation later became grossly impaired, but reaction to convergence was preserved, indicating a lesion in the Edinger-Westphal nucleus. The ocular lesion was complicated by terminal conjunctivitis and exposure keratitis.

Over the latter stage of her illness, the patient developed recurrent urinary tract infections: *Bacterium coli* and *Proteus vulgaris* were cultured from the urine, and her blood urea level rose from 28 to 137 mg. per 100 ml. prior to her death. During the same period she complained of muscle pain on moving her legs, and exhibited signs of peripheral neuritis—foot-drop, absence of reflexes and impaired sensation. In the terminal weeks of her illness many of the skin lesions became ulcerated.

The dosage of prednisone, which had again been elevated to 60 mg. per day without effect, was slowly being reduced when death occurred 88 days after her admission to hospital.

[1] For Figures I to IV see art-paper supplement.

Autopsy.

At autopsy, the positive findings were few. The body was noted to be cachectic with little subcutaneous fat, and was covered with numerous skin ulcers involving the thighs, the arms and elbows and the buttocks. The ulcers were shallow, with black necrotic bases varying in diameter from 1·5 to 5 cm. Other areas of the skin were discoloured with brown pigment and creased as though the tissues beneath had atrophied.

Examination of the cardio-vascular and respiratory systems revealed no abnormality beyond a moderate degree of œdema of the lower lobes of the lungs.

In the alimentary system, the abnormal findings were in the liver, which weighed 2100 grammes and was soft. It was of a yellowish appearance, with small red areas scattered throughout, suggestive of hæmorrhage or necrosis. The spleen weighed 170 grammes and was normal. There was no involvement of any of the internal fat deposits, and examination of the central nervous system revealed no abnormality.

Histopathology.—Examination of a microscopic section of one of the ulcers revealed collapse of the walls of the fat cells, with infiltration by lymphocytes and foamy macrophages. There was a perivascular distribution of lymphocytes and macrophages in some sections.

The liver contained a large amount of fat, only a small number of normal liver cells being seen in a low-power field. A feature was the occurrence of numerous areas of necrotic fat with polymorphs, lymphocytes and plasma cells surrounding them, some of the cells being inside fat spaces. However, the fat-filled macrophages seen in the skin lesions were absent. The inflammatory areas did not bear any constant relationship to the portal tracts or central veins (Figure III).

The muscle fibres showed fragmentation, with vacuolation and necrosis. There was marked cellular infiltration with macrophages, lymphocytes and plasma cells. Some of the macrophages showed densely-staining basophilic inclusions, some of which had "haloes". Some of the vessels were surrounded by collections of lymphocytes and macrophages. Others showed patchy necrosis and infiltration with inflammatory cells, mainly lymphocytes (Figure IV).

In the spleen there was mild congestion, with an increase in the number of plasma cells throughout the pulp.

Examination of several levels of the brain-stem, including the region of the oculomotor and facial nuclei, revealed no abnormality.

The lungs were slightly œdematous and showed no fat emboli such as those described by Kritzler (1941). There was no evidence in the other organs of a focal inflammatory process comparable with that found in the liver. Sections of the myocardium, an appendix epiploica, the kidneys and the thyroid were normal.

Treatment.

As in the other described cases, lack of response to courses of penicillin, streptomycin, sulphonamide and tetracycline was noted here.

There have been a number of reports of the use of steroid therapy in the disease, and the non-specific effect of this group of hormones is borne out in the present case. There was a temporary fall of temperature with an increased sense of well-being (the latter a common result of steroid administration); but new lesions continued to appear, and the disease ultimately progressed in spite of massive dosage of prednisone.

The use of steroids is obviously worthwhile in order to achieve temporary symptomatic relief, but its effect on the course of the illness must be viewed with pessimism. The reported sequelæ of the treatment of rheumatic fever with steroid therapy have been noted in the introduction.

Discussion.

The diagnosis of Weber-Christian disease in the case described was based on the presence of a relapsing febrile illness with nodules in the subcutaneous tissues, biopsy

of which showed panniculitis. A number of other conditions, such as reticulosis, tuberculosis and septicæmia, entered into the differential diagnosis, but were eliminated by the investigations described above. There were, however, a number of features not typical of the disease as originally described. Ulceration of the skin has been regarded as a rarity in previous case reports, but was present in two other fatal cases (Miller and Kritzler, 1943; Friedman, 1945). It raised the possibility of erythema induratum (Bazin's disease), which was rendered unlikely by the failure to demonstrate tuberculosis elsewhere, and by the absence of tubercles and giant cells on histological examination.

Involvement of the central nervous system has been previously noted (Shuman, 1951), but is sufficiently uncommon to merit comment. Facial palsy, oculomotor paresis and peripheral neuritis were observed in this case. The basis for these lesions is conjectural, vasculitis or fat embolism being possible explanations. Neither phenomenon was present on careful examination of the brain-stem sections. The presence of fat emboli in the lung, as described by Kritzler (1941), would have given support to such an explanation, but was again not demonstrable here. Sections of the peripheral nerves were not prepared.

The diffuse fatty infiltration seen in the liver has been described in many of the subjects coming to autopsy. The unusual feature of this case is the presence of considerable cellular infiltrations around areas of necrotic fat. The absence of similar inflammatory change in the other organs minimizes the possibility that terminal septicæmia was the cause. Although the foamy macrophages of the panniculitis were not evident, we suggest that the changes represent a primary inflammation of the fat in the engorged liver, a phenomenon which has not previously been described.

The condition resembled polyarteritis nodosa and dermatomyositis in some of its aspects, suggesting the possibility of a relationship between panniculitis and the collagen diseases. Polyarteritis nodosa was seriously considered during the course of the illness, especially in the terminal stages, when disseminated neurological signs were added to the skin and mucosal lesions already observed, and the presence of inflamed, thrombosed and sometimes necrotic vessels in the skin biopsies and a muscle at post-mortem examination would support such a diagnosis. However, inflammation in vessels and even necrosis is a feature of other collagen diseases, such as dermatomyositis and disseminated lupus erythematosus, and also of severe local inflammation (Plant, 1951; Wallace *et alii*, 1958), and therefore cannot be regarded as specific.

The inflammatory changes in the muscle, together with the skin lesions, suggested dermatomyositis. A relationship between that disorder and Weber-Christian disease was postulated by Walton and Adams in their monograph on polymyositis (1958). The muscle changes described in this case have not previously been reported in panniculitis; although Kennedy and Murphy (1949) described a case in which muscle pain and tenderness were prominent, no muscle biopsy was available. It is interesting to note that 35 years ago Weber and Gray (1924) wrote:

> It is a question whether there may not be minor (incomplete) forms of dermatomyositis in which . . . the muscles appear only very slightly affected, and in which consequently a diagnosis of multiple relapsing panniculitis has to be made.

Although the required pathological changes for the diagnosis of dermatomyositis were present in this case, the typical clinical features — periorbital œdema, erythematous skin, markedly tender muscles and joint changes — were lacking. It appears, therefore, that as some authors [illegible] and Morgan, 1955) have stressed, accurate labelling of a case is [illegible] instances of the collagen diseases depends more on the [illegible] than on the pathologist using [illegible] specific.

The clinical picture, the presence of arterial necrosis and myositis, and the partial response to steroid therapy are features common to collagen diseases, and it is suggested that nodular panniculitis may be yet another variant of this group of disorders.

Summary.

Weber-Christian disease is described and the more recent theories concerning its ætiology are reviewed.

A representative case is discussed, with post-mortem findings. Certain features are reported for the first time, including cellular aggregates and arterial necrosis in muscle.

It is suggested that the clinical picture, the necrosis in vessels of muscle and other tissues, and the partial response to steroids, lends support to the postulate that this is yet another "collagen disease".

It is believed that this is the first case of the generalized disease to be reported in Australia.

Acknowledgements.

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Legends to Illustrations.

FIGURE I.—Photomicrograph from the first skin biopsy showing numerous foamy macrophages and lymphocytes invading the interior of the fat cells. The cell membranes of the latter have ruptured in places. (Hæmatoxylin and eosin stain, × 300.)

FIGURE II.—Photomicrograph from the second skin biopsy showing a thrombosed vessel with an œdematous necrotic wall and a perivascular collection of lymphocytes. Adjacent to the vessel is some inflamed fat. (Hæmatoxylin and eosin stain, × 138.)

FIGURE III.—Photomicrograph of the liver showing a collection of inflammatory cells, mainly lymphocytes, plasma cells and macrophages, but with a few polymorphs in a focus of necrotic fat. (Hæmatoxylin and eosin stain, × 300.)

FIGURE IV.—Photomicrograph of thigh muscle showing degeneration of muscle fibres with a diffuse interstitial inflammatory reaction. A vessel in the lower right corner shows inflammation with necrosis in its wall, as well as a perivascular collection of lymphocytes and polymorphs. (Hæmatoxylin and eosin stain, × 54.)

SOME ASPECTS OF THE HISTORY OF INFANT WELFARE IN SOUTH AUSTRALIA.[1]

By HELEN MAYO, O.B.E., M.D., F.R.A.C.P.,
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THE last quarter of the nineteenth century saw the beginning of organized welfare work for children in South Australia. It was not called infant welfare, as its scope included children of all ages.

Three agencies played an important part in its development, and though they were not aimed directly at the improvement of infant health, all contributed eventually to the lowering of infant mortality rates in the years from 1900 (when the rate was 100 per 1000 live births, with the high figure of 35·52 in the first month of life) to 1910 (when it had fallen to 67·14 per 1000 live births and 26·28 in the first month).

Our welfare work for infants in South Australia began in September, 1909. The agencies referred to were as follows: the establishment in 1872 of the system of boarding out destitute children with licensed foster mothers, of which a woman, Miss Caroline E. Clark, was the pioneer advocate in South Australia, and which subsequently included inspection by the State Children's Department; the opening of the Children's Hospital in 1876, for which Dr. Allan Campbell and his brother were largely responsible; and the passing of the *Public Health Act* of 1898. Before 1909 some beginning of interest in infant welfare elsewhere in Australia had been shown, when Dr. George Armstrong, City Health Officer of Sydney, in 1904 instructed his nurses to weigh babies at regular intervals and give advice to mothers.

[1] Read at a meeting of the South Australian Branch of the British Medical Association on July 30, 1959.

The next notable activity was in New Zealand, where in 1908 Dr. Truby King began to organize what was to become the Plunkett System.

In South Australia we had heard nothing of these ventures, when in May, 1909, an interstate conference of workers for dependent children was held in Adelaide. My friend Miss Harriet Stirling and I were invited to the conference. Miss Stirling was a visitor for the State Children's Department to the homes of children boarded out in her district at Mt. Lofty. She became in 1914 a member of the Council and eventually its chairman.

I had returned from overseas in 1906 to practise in Adelaide. During my medical course at the University of Adelaide I had spent my third year's initiation into clinical work at the Adelaide Children's Hospital, as, owing to the so-called "hospital row", the wards of the Adelaide Hospital were closed to us. On my return from abroad, I was appointed honorary anæsthetist to the Adelaide Children's Hospital. Later I became medical officer to children boarded out with licensed foster mothers by their own mothers. I had had, therefore, some association with children's problems, and, I presume, for this reason I was asked to give a paper on infant mortality at the conference. In reading for this paper I became aware of the upsurge of interest in infant and child life in the world. Budin's *"gouttes de lait"* in Paris and the Huddersfield experiment were evidence of this.

Our statistics showed that the first month of life was the most dangerous to the infant, with an extremely high mortality rate. The first week was even worse. In emphasizing this, I urged that the conference should recommended early notification of births. This was done. However, there was at that time no way of implementing such a law, even if notification was given. It was not until 1928 that the Act was passed.

Later in this year, 1909, Mrs. Napier Birks was visited in Adelaide by her brother, Mr. McDougall, who was a worker in St. Pancras' School for Mothers in London. At a meeting to which Miss Stirling and I were invited, he told of the work that was being done there. We believed that this was what we had been seeking, and we decided to start a school for mothers in Adelaide.

We borrowed the Franklin Street kindergarten for one afternoon a week and engaged a trained nurse to weigh the babies, and Miss Stirling and I attended to give advice. A friend gave us £10. At the end of our first year we were free of debt. This was the beginning of the Mothers and Babies' Health Association of South Australia as you know it today.

At the end of our first year we held an annual meeting, with Dr. A. A. Lendon in the chair. He was most scornful of what we, as spinsters, could teach mothers. This was natural enough, perhaps, because of the dearth of knowledge about the principles of infant feeding, and the physiology of breast feeding. But the ignorance that prevailed among mothers was great. We, at least, knew that breast feeding was life-saving, and that cleanliness was most important. We knew enough to fight against the use of long-tube bottles, and by weighing babies to estimate their growth. We taught hygienic methods of dealing with food, and of preventing infection.

In those early days there were no hospital beds for sick babies aged under two years. The Adelaide Children's Hospital accepted surgical patients even if they were very young, but no medical patients aged under two years. Tiny infants had to be cared for in maternity homes.

We knew that there were babies dying of gastro-enteritis, without anywhere that they could be placed for treatment. Some maternity homes took infants that could not be cared for in their own homes, but none were suited for very serious cases. Among these homes was one called "Quambi", the building on South Terace, now almost derelict, which we have bought to consolidate our land space. In this home was a Nurse Bone, who, although technically untrained, was intelligent and devoted, and would follow instructions with care and diligence. With

her cooperation we learned much, in both the feeding and the management of difficult children.

During a holiday in the eastern States, Miss Stirling and I consulted doctors there about putting sick infants in hospital. With one accord they said: "Don't. You'll lose more by cross-infection than you save." This deterred us for a time. However, the situation grew no easier, so we called a meeting of doctors whose practices included children, and put our problem before them. After discussion they recommended that we should make an investigation to determine its magnitude. Four nursing homes were asked to take in babies, the doctors to attend in an honorary capacity. At the end of a year the need had been proved.

We then decided to approach the Adelaide Children's Hospital to ask if they would build a ward for sick babies in their grounds if we supplied the money. The Board considered the proposition, but eventually declined.

We then began to look for a house in which we could make a beginning, and ultimately rented a two-storey one in St. Peters, engaged staff, appointed honorary medical officers and began. This was during the first World War. Actually, the Adelaide Children's Hospital, shortly after our approach to them, decided to admit sick babies to their wards, but they did not let us know.

We did not have a very easy time in our hospital. It was difficult to get quiet during the day for the night nurses, and at night for the day nurses. There were all sorts of difficulties, but the worst, as we went on, was finance. Therefore, we went to the Government, which, after some consideration, took us over completely and moved the hospital to Woodville, giving it the name of Mareeba. This was in 1917.

The honorary medical officers at Woodville were different, owing to the geographical position. I was appointed Responsible Honorary Medical Officer, and Miss Stirling Secretary. By this time Miss Stirling had had extensive experience. As a member of the State Children's Council she had personal knowledge of the problems of institutions. Together we worked out a plan to prevent, as far as possible, the danger of cross-infection. You are all familiar with the plan nowadays, with a locker for each baby's needs, a gown for the nurse while attending that baby, and so on. It was a help, but not a complete answer.

At Mareeba we, the honorary medical officers concerned, made many experiments in feeding, designed to guide us in our work. On the whole, we were interested in the American ideas, rather than those in vogue in the other States. Mareeba has grown with the years, and has taken part in various special efforts, but I will refer only to one which was undertaken during the latter part of the second World War, in an attempt to lower the death rate of premature babies. It was a plan which had been mentioned by Sir Leonard Parsons in *The Lancet*. It reduced handling of the baby to a minimum. It was a success, in that the death rate of these infants was greatly diminished; but after the war, with the Coronation gift of humid cribs, the picture changed. Thus still further advance was made; the babies were retained in maternity hospitals, away from the possible infection of sick babies, the cribs maintaining warmth and moisture, and oxygen, as found desirable.

This is a digression: I now go back to the period 1909-1919. During these years we learned much about infant feeding. From statistics it was apparent that the chance of life for breast-fed babies was much better than for those artificially fed, but the present understanding of the physiology of the function, its establishment and maintenance, was unknown to us then. Artificial feeding was a controversial subject. Should milk be boiled or not? What dilution was best, and what diluent was the proper one? The merits of various forms were advocated by all and sundry.

A definite step forward was taken when test feeding of breast-fed babies came in. It taught us that the amount taken at each feed varied considerably, and so we might expect an infant on cow's milk to have feeding times of more or less requirement. At a later period there was a move to dilute cow's milk to make it resemble human milk. This was very strong in the Truby King teaching; but it soon appeared to us that real humanizing of cow's milk was not possible, and that the high casein content of cow's milk was well tolerated with certain adjustments. Dr. Truby King went so far as to say that high-protein feeding caused nephritis.

We went through the phases of underfeeding and overfeeding, but finally decided that underfeeding was a serious danger, and that apparent overfeeding on a well-balanced mixture was of little harm. When the "theoretical caloric requirement" was adopted to determine the baby's needs per pound of body weight, it was easier to decide on the possible needs of a baby, but even then there were individual differences. So much is known today that it must seem strange to you that everybody did not know this. It seems odd, too, to realize that at that time only one vitamin had been discovered.

In 1919 there had been a rise of interest in human relations and their significance in child management and in illness, with Sir Arthur Hurst's work on psychotherapy for "shell shock", as it was called, and Dr. H. C. Cameron's book, "The Nervous Child", which was of great importance to our work. The following year I was in England, and was fortunate in being able to attend Dr. Cameron's child clinic at Guy's Hospital, a most interesting experience. Later that year I was at a British Medical Association meeting at Cambridge, when Professor Gowland Hopkins delivered his epoch-making address on the three vitamins, A, B and C. One doctor in the audience exclaimed: "Three vitamins—there will be 73!" Dr. (later Dame) Harriette Chick and Dr. Elsie Dalyell followed, with an account of their work among the starved inhabitants of Vienna. Dr. Dalyell was an Australian, and afterwards practised in Sydney as a pathologist.

One other experience was relative to such work as ours. This was a development, as a side line, of the Liverpool Association for Mothers and Babies; in this Sir Robert Jones used the sisters of the association to keep him in touch with orthopædic patients out on the districts, having given the sisters a short course of instruction. I was interested to see a hospital sister and a welfare worker going round with Dr. McMurray at the Liverpool Children's Hospital, the latter taking notes of the children needing such watching; these notes were sent on to the central offices of the association, and then out to the appropriate district.

In 1921 Dr.—now Sir—Truby King visited Adelaide. We learned about his work and showed him what we were doing. We had been, perhaps, the first in Australia to use lactic acid milk, and we showed him babies who had taken the high proportion of protein in this food, but who showed no signs of nephritis. I think that, perhaps, he may have even given up his idea of such baleful effects, at that time; but it certainly persisted with his trained nurses for some time longer. After his retirement and death, the principles of feeding as taught in New Zealand changed.

The year 1924 was marked by the advent of our first pædiatrician, Dr. F. N. le Messurier, who returned from America. He brought back the idea that gastro-enteritis was often associated with otitis media and mastoid disease. On the whole, we were rather doubtful, but when we took to examining ears in all cases we changed our minds. Actually, this seemed for some time to be a common occurrence; but later the picture changed, and otitis media appeared to be a specific infection, present only in some epidemics. However, a child with severe gastro-enteritis, whose life was saved at the St. Peters hospital, was found later to be completely deaf, with evidence of past otitis, suggesting its association with the gastro-enteritis attack some years before.

In 1933 I went to London to attend the International Pædiatric Congress. Dr. le Messurier was there also. There was much to interest us, particularly a demonstration by Dr. Samuel Karelitz of the intravenous drip method. Here was another step forward in the treatment

of disease in infants and others, leading on to the understanding of electrolyte balance, and to much greater knowledge of the constitution of the body fluids, and of how to correct their abnormalities. Besides this, it encouraged the use of blood transfusion, and led to the recognition of the dangers of incompatible blood—and, further, of the great danger to the unborn child of incompatible blood in the parents. This knowledge has led to the saving of many lives in the neonatal period.

Since the year 1924, when Dr. le Messurier returned from America, infant mortality had been slowly decreasing—*post hoc* or *propter hoc*? In 1925 it dropped to 46·89, with first-month deaths 26·71. Although in 1927 it rose to 53·43 and 30·63, it has never since touched the fifties.

In several years South Australia achieved the lowest mortality rate in the Commonwealth, and on more than one occasion a world record. The lowest rate so far was in 1957, with 20·63 per 1000 live births, and 13·77 in the first month, possibly a world record.

It is generally conceded that the consistent downward trend of the State's infant mortality rate (it was halved during the first 20 years of the Association's existence) has been due very largely to the work of the Mothers and Babies' Health Association, which has grown from its humble beginnings as the School for Mothers (open one afternoon a week in a borrowed kindergarten) to the organization which today conducts more than 240 baby health centres throughout the State, runs three baby health trains to areas where are no centres, and conducts Torrens House and a variety of other services to the community in maternal and infant welfare.

The establishment of a residential training school for sisters in infant welfare and for mothercraft trainees, with accommodation also for mothers and babies, was recognized early in the 1920's as essential to the full development of our objectives.

It was difficult, for instance, to establish, or to reestablish, breast feeding unless the mother was in an institution where such ideas were accepted as a principle. Such institutions existed elsewhere in Australia; but for various reasons, including the financial depression which began in 1930, we did not achieve this objective in Adelaide until 1938, when Torrens House was opened.

Our annual expenditure today is more than £80,000 and our fixed assets are more than £156,000. Our finances have been helped greatly by the State Government, which gave us £50,000 last year and is most generous to us.

The Association has had three medical directors, Dr. Elma Sandford-Morgan, who was appointed part-time in 1938, Dr. Ruth Mocatta, who succeeded her in 1941, and Dr. David Fearon, first full-time director, who was appointed in 1957. Since 1932, the Association has had a Medical Advisory Board, consisting of representatives of institutions dealing with infant and maternal care, to help in guiding its medical policy, and in developing cooperation between medical practitioners and the baby health centres.

Any reference to the work of the Mothers and Babies' Health Association would be utterly inadequate without reference to the thousands of voluntary workers, including a number of doctors—they may be called "the unseen hosts"—who have given devoted service to the Association during all the years of its existence, from its very beginning.

I have given you but a slight sketch of a period that really extends over nearly 90 years, although my own association with these events covers but 50. How tremendous have been the changes during that period! How different is our world from that in which I grew up!

In our work, though our aims were the physical, mental and social well-being of mother and child, we necessarily placed varying emphasis on the needs of the moment. Thus the first 10 years were devoted largely to nutrition, and to the provision of institutions in which to deal with serious illness in infants. After this period, the emphasis was on human relations in association with illness and emotional disturbances—an important aspect of a problem on which much remains to be done. Another thread in the whole pattern was the arrival of the first pædiatrician, and the beginning of this specialization. The work of the Association for the next few years—indeed, until now—was concerned with the growth of centres, the increase of staff, the opening of our training centre, Torrens House, teaching by means of publications and lectures to schoolgirls, the correspondence department, the financing of all these material advances and, last, but by no means least, the labours of the sisters with the mothers and babies.

Meanwhile, in the world of medicine and pharmacy, the discoveries of the sulphonamides and the antibiotics have changed the prognosis of many infections, and have, besides, introduced fresh problems. The demonstration of the intravenous drip method did this also, and with its associated blood transfusions led to further specialities, and to the recognition of blood incompatibilities and their alleviation and to the understanding of many states of health and disease formerly only partly known. Nor, in referring to the advances in the fight against a high infant mortality, should I omit those that have been reached through advances in the surgery of the infant. The operation for hypertrophic pyloric stenosis was one of the earliest; but we have lived to hear of many more procedures involving the heart, and hitherto unapproachable organs, with the undoubted saving of lives that might otherwise have been forfeit.

The first week is still a dangerous time for the new life, but the first month has become less so, and we hope that by help to the expectant mother in nutrition and in other ways that may become more evident, we shall go even further in our warfare against the dangers that beset the very young.

We see some directions in which we want to develop. These include the training of sisters, and the means we can adopt to keep their knowledge up to date and their part in the whole organization an active one. Closely interwoven with our plans is our desire to bring into the Association the general practitioners, whose help is all-important in such problems as those of family life.

In our early days we were not liked by doctors as a rule. They believed that we were trying to rob them of their patients. We think and hope that the medical students of today feel differently about this. The opportunity of seeing something of our teaching at Torrens House, Mareeba and the Adelaide Children's Hospital has changed the attitude of future doctors to problems of infant welfare, and we hope will continue to do so more effectively. Our sisters do try to enlist the help of the family doctors in charge of patients, and not to interfere.

After the years of the second World War, we found that the medical students, who were more mature than the ordinary students, were immensely interested in problems concerning children. Many of them were married and had children of their own.

Our greatest difficulty is to meet our need for sisters. Nurses are now so travel-minded that the idea of settling down to a steady job does not appeal to them until they have seen the world. Besides, there is the constant wastage, as it is called, from the profession by marriage, which deprives us of many splendid sisters, although we should not regret their choice of another important field of service.

I have touched only lightly on the problem of human relations. It is, however, of first importance, not only to us, but to the whole world. Parents—fathers as well as mothers—doctors and sisters, social workers and many other people need to be coordinated to help to create and maintain the stability of that most important unit in our society, the family.

This concludes my sketch of the history of infant welfare in South Australia, with some indication of the growth of knowledge in the scientific world at a rate unparalleled in our civilization. If this 50 years has brought us so far, leaving many problems still to be tackled, what will the next 50 years bring?

INFANT WELFARE IN SOUTH AUSTRALIA TODAY.[1]

By DAVID FEARON,
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IT is a particular honour to follow Dr. Mayo — and a responsibility. Almost fifty years have passed since the School for Mothers was started by her; but the vision of the idea is not lessened by time. All that is needed in planning the next fifty years is this same inspiration, with a reassessment of the needs of parents and children and planning to meet them. In effect, the idea of a school should still be in our minds; the curriculum and content of its teaching must be up to date and relevant to its pupils' needs, and its teaching should incorporate modern educational methods.

I have been asked to say something of the Mothers and Babies' Health Association today — what it is, what it does, and how it does it. I shall also say something of my own observations and impressions of work in this field.

The executive and policy-making body of the Association is the Central Committee. Branches are formed primarily for the purpose of raising money towards the local health centres. The Central Committee consists of 19 interested women, a number of whom have had many years' experience in its activities. Provision is made in the constitution for two representatives of the branches. The Central Committee has five subcommittees to deal with details of the Association's work.

Advice on medical aspects of policy is given by a Medical Council composed of appointed representatives of bodies and institutions concerned with the care of mothers and children. They are the South Australian Government, the British Medical Association, the Department of Obstetrics, University of Adelaide, the Mareeba Baby Hospital, the Adelaide Children's Hospital, the Queen Victoria Maternity Hospital (two representatives), and the Queen Elizabeth Hospital.

Staff.

A clerical and book-keeping staff is employed at headquarters under the direction of the State Secretary of the Association.

Medical and Nursing Staff.

The Medical Director is responsible to the Central Committee. Duties include supervision of the activities of the nursing staff at headquarters and in health centres, with supervision of the care of mothers and babies admitted to the mothercraft home, Torrens House, and of the training of infant welfare sisters and mothercraft nurses. The Medical Director is also Tutor in Mothercraft to the University of Adelaide. Medical students make a number of visits to headquarters and to Torrens House in the last two years of their course, to learn something of the mothering of children and of health-centre work.

Other members of the headquarters staff are the matron of the health centre staff and the assistant matron. The correspondence sister's work in helping those out of reach of our clinics has been mentioned, as has the work of the two lecturing sisters, who teach mothercraft in 21 metropolitan girls' schools. (Classes are also held in some country high schools.)

At headquarters, lectures to expectant mothers are given by the matron, and a physiotherapist conducts prenatal relaxation and exercise classes.

The nursing staff in the health centres are all triple-certificated sisters, 40 full-time and seven part-time.

Torrens House.

This is the only mothercraft home in the State. It is staffed by a matron, who will have an assistant when a suitable person is available, and three full-time sisters and one part-time sister.

The following types of patients are admitted to Torrens House: eight newborn babies with their mothers, when problems of management and feeding occur in the neonatal period; ten babies alone — again for problems of management and observation; three toddlers up to the age of two years, for disturbances of behaviour, sleeping, feeding, etc.

The student nurses are of two types — those doing the third certificate in nursing in Australia, infant welfare (here a four-month course) and trainee mothercraft nurses. These latter are previously untrained girls doing a twelve-month course.

Disappointingly few of the infant welfare trainees enter our own health centre service. Some mothercraft nurses take cases in private homes on completion of training.

It is planned to build a new mothercraft home on the large site on South Terrace now owned by the Association. On this site it is hoped to bring headquarters, community service and training activities closer together.

Work of the Centres.

Last year, nearly 70% of the babies born in the State were enrolled at Mothers and Babies' Health Association health centres. Many more were visited in their homes. The average number of attendances per baby was between seven and eight.

Great distances and sparsely populated areas are constant problems in provision of service. Health centres are conducted in 240 places. Of these, 99 are in the metropolitan area of Adelaide; of the remainder in country areas, 43 are serviced by the three baby-health trains. These are converted carriages, one end waiting and consulting sections, the other the sister's living quarters. Most of the other country centres are visited by a member of the staff by car from a central point in the district.

Most of us know something of what is done in an infant welfare or baby health centre. I should emphasize four aspects of this work.

Feeding Advice.

Food is important; but advice on meeting the baby's need for food, if well given, introduces the mother to most of the basic concepts of child care. It can exemplify and tie in with the meeting of the child's needs in many other areas. One does not need to see many mothers and babies to be impressed by the continuing need for guidance on elementary principles.

Weighing and Observation of Babies.

The grossly underfed baby, breast-fed or bottle-fed, who does not complain, or whose behaviour is not understood, remains the justifying example of the need for routine weighing. Abnormalities and disease processes are often first suspected by the detached and experienced observations of a good clinic sister. She may note a change in appearance or behaviour too gradual for the baby's immediate family to have observed.

Support of the Mother.

There is a significant amount of anxiety which can be dealt with at the symptomatic level. The nurse working in the centre has a very real and important role in reducing maternal anxiety. However, to encourage dependence threatens the future of the service, the development of the mother's ability and, worst of all, the future of the baby. Health education will do much to encourage the independence of mothers. However, no book or lecture will replace kindly understanding of an individual mother's difficulties with her baby.

Relations with the Family Doctor.

The doctor is, and must remain, in charge of the medical care of the mother and child. I wish to be

[1] Read at a meeting of the South Australian Branch of the British Medical Association on July 30, 1959.

quite clear. All the staff understands that nothing must be said or done which may adversely affect this relationship. From time to time, reports of nonconformity with this rule are received. In almost all cases I have found them to be the result of misunderstanding or misquotation. Such matters are best reported and investigated—from everyone's point of view. If the doctor wishes some line of management taken with a baby, the sister will follow his lead; but I am afraid she does not always receive it.

One of the worst things that can happen to a mother, and hence to her baby, is to receive conflicting advice. Recognizing this, some doctors, to avoid it, discourage clinic attendance. If a mother is regularly attending a physician with her baby, I should rather she did not attend a health centre than that she there received conflicting advice; but it does seem a pity that she should be denied help that she might get because of failure of communication between two people, both of whom are potentially important to her.

Some Personal Observations and Impressions.

We have just had a most unusual and valuable experience. We have heard, in a few brief minutes, the outline of fifty years' work for mothers and children, encompassed by the memory of one worker in this field, Dr. Mayo. It is hard, probably impossible, for us who did not have Dr. Mayo's experience to really appreciate the state of affairs that existed in the care of young children fifty years ago. To us now, in retrospect, the needs in the community of that day seem clear, and the service established to meet them well planned.

What are the needs in infant welfare today? How, in this day of antibiotics, mass immunization and endocrine replacement therapy and other very technical medical knowledge, can one evaluate this type of work? Of necessity, workers in their field fifty years ago had to concern themselves primarily with the physical needs of babies. And yet many of them, Dr. Mayo in particular, were not unmindful of the whole of the child's life.

Today, as the result of the efforts of workers in many fields, we live in a community with high standards of maternal and infant care. The risks of reproduction to the mother are small, the chances of a live birth are excellent, and the possibility of survival by the infant for his first year of life is about fifty to one.

There is still much to be done on the physical side. A great part of it will be achieved by technically skilled workers, doctors and associated scientists. But there are still physically unhealthy children, and diseases which we know to be preventible still occur in large numbers and have an appreciable death rate; a check on admissions to children's hospitals and a study of the list of causes of death in babies prove this.

If we make more information available to parents, we should reduce preventible disease and deaths; but to improve results, the methods of presentation will become increasingly important. Many of the parents of these children are not just ignorant or wilfully negligent.

If this is true of meeting physical needs, and I believe it to be, it is of even greater importance in teaching parents to meet the emotional needs of children. If a mother's physical and psychological preparedness for breast feeding is inadequate, it is absurd and harmful to tell her that it is her duty to breast feed and she must try. Similarly, if there is subconscious hostility to a child, the constant urging of understanding of his needs and telling the mother to show him more love do more harm than good. She needs help, trained help, to understand and vary her attitude.

Some parents find it difficult to give children the things they need; others find it difficult not to give them things they do not need. The problem is then not ignorance, but attitudes born of emotional difficulties within the parents themselves.

In brief, we must work not only to make more information available, but also to help parents to use this information. It is for this very purpose that recommendations are repeatedly made that more adequately trained personnel are needed — people who understand the psychology of human relations, who have studied and are aware of subconscious drives and have skill in directing them.

We are realizing more and more that health involves the whole person. Primarily, this service is planned to meet physical needs, and so by good physical care and diet save the babies and aid their future. We are now faced with a challenge. How can we improve the quality of the lives of the survivors? In these stressful times emotional maturity is of increasing importance in the lives of individuals and nations. We are becoming more aware of the part played by our feelings and circumstances in a wide variety of diseases previously thought of as purely physical. Much mental ill health and some mental diseases, it is held, are related to events early in childhood. Increasing attention is focused on the first years of life. Are there problems of individual, family and community health which we might attack earlier?

Recently, a senior policeman in London was reported to have stated that neither the absence of real poverty nor the employment of up-to-date methods of dealing with juvenile delinquency and crime seemed to have had any effect on the problem.

It has been put to me that our work should be concerned with the promotion of parenthood as a vocation. This is not something for which one can legislate, or on which one can give directions. It is a matter for encouragement by a variety of means. Classes and discussion groups for parents and prospective parents may help. Some of our branches have started mothers' clubs—that may be a beginning. In New Zealand, the rise in popularity and strength of the Associated Parents' Centres is a development of great interest.

I believe we should encourage community participation in health education.

But for these activities to be effective, more trained help is needed. We need more staff very urgently, to maintain a good service to individual mothers even at the present level. Their training should be under constant review in the light of changing community needs. For activities such as those suggested and to meet the broader aims we need a vigorous, closely knit organization and much more trained help. Will the medical profession help us in what I believe is sound preventive medicine?

Dr. Mayo has, in effect, made a report and invited suggestions for the future. May I, in closing, summarize the potential of this service and organization? More than 70% of mothers of all babies born are contacted (for first-borns the percentage is probably much higher). We run the service and also the training of those who give it. We are not bound by a mass of government departmental regulations. Throughout the community we have this large body of interested supporters.

EARLY DIAGNOSIS OF CARCINOMA OF THE CERVIX BY COLPOSCOPY AND CYTOLOGY.

By R. FRANCIS MATTERS, M.S., M.D., F.R.C.S. (Edinburgh), F.R.A.C.S., F.R.C.O.G.,
Adelaide.

FOR some years I have used the Papanicoloau smear test and the colposcope in the diagnosis of cervical carcinoma. These methods have supplemented pelvic examination by digital methods and visual inspection by means of a vaginal speculum, and also the use of Schiller's iodine test.

After an extensive visit to most large North American clinics, and also those of the British Isles and of Europe, a comparative estimate of diagnostic results has been made.

The most complete cytological diagnostic centre which was seen is directed by Ruth Graham, of Buffalo.

J. V. Meigs, of the Massachusetts General Hospital, has had a great number of cytological diagnoses followed by examination of serial sections of the cervix; this was also done by Te Linde, of the Johns Hopkins Hospital, Baltimore.

At Cornell University, New York City, Professor Papanicoloau takes the vaginal smear by aspirating from the pool behind the cervix with a pipette. The aspirated fluid is then smeared on a slide placed in a mixture of equal parts of ether and alcohol for fixation before staining.

The method adopted by us follows the technique of Navratil of Graz, which is a slight modification of Ayre's method. This involves the taking of a smear on a dressed swab stick, and in addition using a tapered wooden spatula which, by a rotating motion, scrapes the mucocutaneous junction of the cervix. In addition, we now aspirate the vaginal pool. The material obtained is smeared on a slide and, after fixation and staining, is examined by a cytologist.

Lindberg, who followed Hinselmann in Hamburg, makes a colposcopic examination of every female patient, but this is not necessarily associated with the taking of the smear.

Ernst Navratil has a large clinic, the Graz University Frauenklinik, where every patient has both cytological examination and colposcopic investigation. Navratil considers that the simultaneous use of colposcopy and cytology requires little more time than does speculum examination, and if a biopsy is to be taken, the exact spot may be chosen by colposcopy. Many biopsies are found to be unnecessary after these examinations. Navratil states that the purpose of all available screening methods used in the diagnosis of carcinoma of the cervix is the detection of the early stages of the disease, which otherwise escape recognition by the simple methods of clinical examination, such as palpation, visualization, etc.

The accuracy of cytology when used as a screening procedure in the detection of preclinical cancer, which is asymptomatic and corresponds histologically to early invasive cancer, is usually quoted in the literature as between 70% and 90%. Ruth Graham's figure of 86% should be a very good standard of thorough screening, because of the great cytological experience involved.

Navratil states that, in view of the importance of early diagnosis to the results of treatment, it is imperative to find a way to improve our diagnostic accuracy without resorting to multiple reexamination. Patients resent frequent examinations even in well-organized clinics, and to this is added the fear of a positive finding. Other methods should be used at the time when the smear is taken; these include Schiller's iodine test, biopsy from suspicious areas and the colposcopic examination, which makes it simpler to take a biopsy from the right area. With the naked eye, through the speculum, patches of leucoplakia may give a false impression.

With regard to biopsies, it has been shown that in less than 50% of cases of intraepithelial carcinoma were positive findings obtained at the first biopsy. However, Graham and Meigs found that in 40 cases the original biopsy resulted in 70% correct diagnoses; but the great knowledge and experience of these workers were able to produce these figures.

Four-point biopsies have been indicated by Foote and Stewart; these are taken at the 3, 6, 9 and 12 o'clock positions on the cervix. Ayre prefers conization of the cervix, but this becomes an operative procedure.

Schiller's test with Lugol's iodine is most helpful in determining the site of biopsy. However, none of these methods gives the extra advantage obtainable with the colposcope.

Once more to quote Navratil, the control of errors in a specific method can be achieved, by the use of another specific screening method; such a method is colposcopy in accordance with Hinselmann's technique.

Antoine of Vienna uses even greater magnification of the epithelial cells of the cervix; but this method is a longer and more tedious method for the patient, without a greatly improved diagnostic result.

Conclusion.

After seeing so many clinics during the past four months, I believe that the simplicity of colposcopic examination and its indication for target punch biopsies increase our early diagnostic methods quite appreciably.

In conjunction with cytology, colposcopy is very useful in the early diagnosis of preclinical carcinoma of the cervix and allows the investigator greater accuracy in making a punch biopsy.

It is considered that every available method should be used in the early diagnosis of cancer. The addition of colposcopy should increase the number of early diagnoses of carcinoma of the cervix.

Reports of Cases.

CHLORPROMAZINE IN THE MANAGEMENT OF TETANUS: REPORT OF A CASE.

By M. K. BENJAMIN,[1]

From the Clinical Research Unit of the Royal Melbourne Hospital and the Walter and Eliza Hall Institute of Medical Research, Melbourne.

CHLORPROMAZINE has been shown to counteract muscle spasm in experimental tetanus (Laurence and Webster, 1958). Adams *et alii* (1959) reported that chlorpromazine given intramuscularly controlled spasms in human tetanus, although the mortality rate reported by them for severe cases was high—50%. The present case report illustrates the usefulness of chlorpromazine administered intravenously in the treatment of moderately severe tetanus.

Clinical Record.

A female patient, aged 44 years, was admitted to the Royal Melbourne Hospital on December 16, 1958, suffering from moderately severe tetanus. Eight days previously a splinter had penetrated her left great toe and transient inflammation occurred. She had not received active or passive immunization against tetanus. Twenty-four hours previously she had noticed tightness of the jaw and difficulty in chewing, and subsequently increasing stiffness of the limbs, tightness of the neck, and severe pain in the lumbar muscles. These symptoms became worse, but spasms did not occur. She could swallow fluids, but a constricting feeling in the chest made breathing difficult.

Examination of the patient revealed trismus, risus sardonicus, rigidity of abdominal muscles, stiffness of the left leg and pronounced opisthotonos. The respiratory excursion of the thorax was limited. The patient's temperature was 37° C. and her pulse rate 120 per minute. Lumbar puncture produced a severe spasm of the trunk and limbs.

Immediate measures included tracheotomy, amputation of the infected toe, and administration of 60,000 units of anti-tetanic serum. A plan of over-all management and nursing care was undertaken as described by Last and Nicholas (1956), by Wilson *et alii* (1956) and by Martin (1957). For the first 72 hours, sodium phenobarbitone (200 mg. every eight hours) and chlorpromazine (50 mg. every six hours) were administered intramuscularly. However, this regime failed to control the disease. At no time was respiration impaired, but generalized muscle spasms occurred, particularly after tracheal aspiration.

The variability of response to intermittent intramuscular injection of chlorpromazine prompted its continuous intra-

[1] Drug Houses of Australia Fellow to the Clinical Research Unit.

venous administration, which was successfully maintained for the subsequent 14 days. A constant infusion of chlorpromazine was given, diluted in glucose and glucose-saline solutions, in dosages of 200 mg. per 24 hours. Thereafter the patient's management became greatly simplified; she remained fully conscious and could report the onset of rigidity, muscle pain and respiratory distress. Moreover, she could fully cooperate in nursing and physiotherapeutic measures. Severe spasms, which were caused by anoxia due to bronchial obstruction by mucus or tracheotomy toilet, were controlled by an additional 50 mg. of chlorpromazine injected into the intravenous delivery tube.

On the seventeenth day chlorpromazine was given orally (25 mg. every three hours), with occasional intravenous doses when mild spasms occurred. On the twenty-eighth day all therapy was discontinued. After recovery had occurred, the patient had no clear recollections of her ordeal.

She had remained afebrile and free of pulmonary complications throughout. There was persistent tachycardia, but no electrocardiographic evidence of myocardial disease. Laboratory tests showed that neither hepatic damage nor bone-marrow depression had resulted from the use of chlorpromazine.

Discussion.

This patient was regarded as having moderately severe tetanus because the incubation period was short (eight days) and the onset of the disease rapid (24 hours), and because reflex spasms and opisthotonos occurred. Continuous intravenous administration of chlorpromazine proved of great value in that it controlled muscle spasm, provided adequate relaxation and allayed anxiety; similar findings have been reported by Cole and Robertson (1955). The oral and intramuscular administration of the drug was much less effective in providing even control of spasms. Medical and nursing care was thus greatly simplified, and the patient remained sufficiently alert to give warning of new symptoms and impending spasms; moreover, a closer contact was maintained with the patient than is possible with alternative forms of treatment, such as curarization and aided respiration. It must be emphasized, however, that curarizing agents, associated with either intermittent positive-pressure respiration or tank respiration, will still be necessary in the severe cases of tetanus.

There is experimental evidence that acetylpromazine may be ten times as effective as chlorpromazine (Laurence and Webster, 1958); if this is so, acetylpromazine may be the drug of choice in severe tetanus. However, Adams *et alii* (1959) could not confirm that acetylpromazine was more effective than chlorpromazine in clinical tetanus. The final place of chlorpromazine and its derivatives in tetanus is thus uncertain and will depend on further clinical experience in the treatment of this disease.

Summary.

A woman with moderately severe tetanus was successfully treated by continuous intravenous administration of chlorpromazine and by tracheotomy. Continuous administration of the drug by the intravenous route was found to be much more effective than intermittent oral administration.

The patient remained alert and cooperative throughout; medical and nursing procedures were thus greatly simplified.

The place of chlorpromazine given intravenously in severe tetanus requires further evaluation; deeper sedation, muscle relaxants and aided respiration will still be necessary in some cases.

Acknowledgements.

I am indebted to Dr. Ian Wood for allowing this case to be reported, to Dr. Ian Mackay for his assistance, to Sister J. James, who supervised the nursing care, and to the staff.

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ORNITHOSIS: REPORT OF FIVE CASES.

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THE purpose of this report is to draw attention to the fact that five patients suffering from ornithosis have been seen in the Royal Newcastle Hospital in the one year, three in the space of one fortnight.

Case I.

A male patient, aged 53 years, from Booragul, N.S.W., was admitted to the hospital on April 18, 1958, with a history of cough, fever and headache of five days' duration. He had been delirious at night for two days. On admission, the patient was confused and slightly cyanosed. His oral temperature was 100·8° F., his pulse rate 96 per minute, and his respiratory rate 32 per minute. Râles were heard in the right axilla and in the right mammary area. On X-ray examination of the chest on April 18, "a lesion several centimetres across in the right lung inferiorly is most likely inflammatory consolidation in the middle lobe" was reported. A sputum culture on blood agar made on April 19, 1958, yielded *Streptococcus viridans* and *Neisseria catarrhalis*. A blood culture was taken on the same day; this was sterile after 10 days. The patient was less well on this day, his temperature was 102·8° F. and his pulse rate 104 per minute, but his respiratory rate was only 20 per minute. Chloramphenicol therapy (500 mg. every four hours) was then begun as the diagnosis of staphylococcal pneumonia with septicæmia was entertained.

The patient gradually improved and was able to be discharged from hospital on May 3, 1958, but prolonged convalescence was needed, and he was unfit to return to work—which was specially arranged to be light and sedentary—until July 15, 1958. A feature of his convalescence was the patient's vivid recollection of distressing nightmares during the acute phase of his illness.

As the patient had bought parrots which sickened and died prior to his illness, arrangements were made for the estimation in his serum of complement-fixing antibodies to the psittacosis virus. A titre of 1:20 on April 30, 1958 (seventeenth day of illness), which rose to 1:40 on May 20 (thirty-seventh day of illness), was obtained.

Case II.

A female patient, aged 37 years, from Cardiff, N.S.W., was admitted to hospital on December 20, 1958. Five days prior to her admission she had suffered from aching limbs and diarrhœa for two days, followed by anorexia, headache, chills and a "funny feeling" in her chest on deep inspiration, together with an irritation in her throat.

On examination, she appeared lethargic. Her oral temperature was 100·4° F., her pulse rate 120 per minute,

and her respiratory rate 20 per minute. Examination of her urine showed a trace of sugar and acetone. No other abnormality was found on physical examination. However, three days after her admission to hospital the tip of the spleen became palpable.

This woman's illness lasted for three weeks in hospital and was characterized by a feeling of extreme malaise. After her discharge there was a recurrence of symptoms lasting several days. No antibiotics were administered during her stay in hospital in view of the provisional diagnosis of psittacosis.

X-ray examination of the chest on December 22 showed "an abnormality on the right side at the level of the hilum, most likely an inflammatory lesion". It was reported after a further examination on January 6, 1959, that "there may be an inflammatory lesion in the base of the right lung".

On examination of the patient's blood on December 22, the hæmoglobin value was estimated at 14·3 grammes per 100 ml. and the white cells numbered 4200 per cubic millimetre. Cold agglutinins were not found. On January 6, 1959, the white cell count was 6000 per cubic millimetre, 64% being neutrophils, 23% small lymphocytes, 5% large lymphocytes and 8% monocytes.

A stool culture on December 24, 1958, yielded no pathogens.

The serum titre of complement-fixing antibodies to the psittacosis virus was less than 1: 32 on December 23, 1958, but greater than 1:192 on January 27, 1959.

The patient has kept budgerigars for years. Some young birds had become ill and died a few days before the onset of her symptoms.

Case III.

A female patient, aged 20 years, from Cardiff, N.S.W., was admitted to hospital on January 3, 1959, with a history of anorexia, vague upper abdominal pain and intermittent vomiting of two weeks' duration. She had developed a dry cough one week before her admission to hospital, and a sore throat and headache on the day of her admission.

On examination, she was in no distress. Her oral temperature was 102·6° F. Her pulse rate was 98 per minute, and her respiratory rate 20 per minute. Examination of her urine revealed no abnormality. The only abnormal physical finding was consolidation in the base of the left lung. Her malaise persisted for one week, as did her elevation of temperature; then she made a full recovery. There was a relative bradycardia throughout her illness. The patient's respiratory rate remained low at 20 per minute throughout. A chest X-ray examination on January 6, 1959, showed "a lesion on the left side, probably extensive inflammatory consolidation in the base of the lung". A further X-ray examination of the chest on January 13 gave normal results.

On examination of the patient's blood on January 8, the hæmoglobin value was estimated at 12·6 grammes per 100 ml.; the white cell count was 4650 per cubic millimetre, of which 82% were neutrophils, 17% were small lymphocytes, and 1% were eosinophils.

The patient was treated symptomatically only.

Serum complement-fixing antibodies to psittacosis were found to be present to a titre greater than 1:192 on January 7, 1959, and again on January 27.

This girl, a neighbour and friend of the woman described in Case II, also kept a parrot on the back veranda, where she slept. The bird had died a few days prior to the onset of her illness and another newly purchased bird, put into the same cage, also died two weeks later.

Case IV.

A male patient, aged 46 years, from Charlestown, N.S.W., was admitted to hospital on December 23, 1958, with a history of headache, anorexia and malaise of two weeks' duration. He had had a cough for three days and profuse sweating the evening before his admission. On examination, his temperature was 101·4° F., his pulse rate 88 per minute, and his respiratory rate 20 per minute. There were no physical signs.

Four days after his admission, conjunctival injection was noted, and about this time the patient's cough increased and he complained of soreness in the left mammary region. His temperature fell to normal by lysis two weeks after his admission, and his recovery was uneventful. No antibiotics were given.

X-ray examination of the patient's chest on December 24 showed doubtful evidence of an inflammatory lesion in the left lung, situated inferiorly.

The total white cell count was 4500 per cubic millimetre, of which neutrophils comprised 74%, small lymphocytes 18%, large lymphocytes 1% and monocytes 7%.

Serum taken for examination on January 27, 1959, six weeks after the onset of the illness, showed complement-fixing antibodies present in a dilution of 1 in 128.

This patient had had contact with sick budgerigars prior to this illness, and during his stay in hospital his landlady's parrot died.

Case V.

A male patient, aged 61 years, from Nelson Bay, N.S.W., was admitted to hospital on January 21, 1959, with a history of aches and pains in the limbs and abdomen, together with a severe headache, for six days. He had a slight dry cough. In the previous month six young parrots he had recently purchased had died, although two older birds had remained well.

On his admission to hospital the patient had a temperature of 102·6° F., his pulse rate was 94 per minute, and his respiratory rate was 26 per minute. The patient looked rather ill, and there was an area of bronchial breathing heard medial to the right scapula. He appeared to be in some distress from his headache, but otherwise physical examination revealed no abnormality. A culture of a throat swab yielded a moderate growth of *Staphylococcus aureus* which was sensitive to penicillin, and in addition a *Bacillus subtilis*. Examination of the urine showed a moderate cloud of albumin and the deposit showed a few hyaline and granular casts, with 10 red blood cells and two pus cells per field. The urine was sterile. The white cell count was 7700 per cubic millimetre, with 77% neutrophils, 10% small lymphocytes and 13% large lymphocytes. No cold agglutinins were detected in his serum. On X-ray examination of the patient's chest, it was reported that "a lesion on the right side is most likely inflammatory consolidation in much of the upper lobe".

A further white cell count on February 11, 1959, showed a total count of 7800 per cubic millimetre, 66% being neutrophils, 15% small lymphocytes, 7% large lymphocytes, 5% monocytes, 5% eosinophils and 2% basophils.

In view of the provisional diagnosis of psittacosis, no antibiotics were administered, and the patient was given symptomatic treatment. He suffered from intermittent rise in temperature up to 102° F. for two weeks, and then his temperature settled to normal by lysis over the next two weeks. Throughout the illness his sole complaints were of headache, anorexia and a slight unproductive cough.

Further X-ray examinations of the chest showed progressive diminution in the radiological changes, so that by February 17 it was reported that "there is now little evidence of the lesion in the right lung".

Serum tests made on February 10, three weeks after the onset of symptoms, showed complement-fixing antibodies to the psittacosis antigen present in a dilution of 1 in 48. A further specimen of serum taken on March 2, six weeks after the onset of symptoms, had these antibodies present in a dilution greater than 1:128, and a third specimen taken on March 17 gave a similar result.

Comments on Serological Findings.

As we have no information regarding the titre of psittacosis complement-fixing antibodies in the local population, it is somewhat difficult to draw definite con-

clusions as to the significance of the serological findings in these cases. It seems probable, however, that the situation here is not greatly different from that reported by Dane in Adelaide, and by other observers in other parts of the world.

The serological findings in Case I suggest past or present psittacosis; in Case II they are probably diagnostic; in Case III, in which the first serum specimen was not obtained until 18 days after the onset of illness, they are very suggestive and probably not due to an anamnestic rise; in Case IV, the titre of the one specimen suggests past or present illness, although the titre is rather higher than might be expected if it was due to past illness; in Case V they appear to be diagnostic of psittacosis.

Acknowledgement.

We are indebted to Dr. Ian Jack of the Virus Laboratory, Royal Children's Hospital, Melbourne, who carried out the serological investigations on these patients.

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TOTAL BLINDNESS FROM TEMPORAL ARTERITIS.

By HUGH RYAN, F.R.C.S.,
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TEMPORAL ARTERITIS, since it was first reported by Hutchinson in 1890, has tended to be regarded as a bizarre and often irritating condition, but without severe complications. This is not so, and the following case is reported to illustrate the disastrous sequelæ which may occur from complications of this disease.

Clinical Record.

Mrs. A., aged 68 years, was admitted to St. Vincent's Hospital, Melbourne, complaining of complete blindess in the left eye of two weeks' duration. She said that she had been well until three months previously, when she had developed pains in the lower limbs. One month later she had developed severe pain in the back between the shoulder blades and around the chest. She described the pain as agonizing. The pains lasted three to four weeks, and gradually subsided. Six weeks before her admission to hospital she had developed similar pain in the throat and the lower part of the chest. This pain lasted for two weeks, and was followed shortly afterwards by severe pain all over the face and chest. This pain radiated to the head. At this time she developed extremely tender patches on the head and face. These patches were mainly around the temples, and there was a small raised patch on the scalp near the vertex. She said that at this time "veins" of the head were hard and raised and sore to touch. Three weeks before her admission to hospital the right eye became suddenly blind; a few days later the vision of the left eye began to fail, and that eye also was blind three days later. The pain had subsided about one week prior to her admission to hospital. The patient said that at the onset of her trouble she had painful cramps in her calf muscles and in the muscles of her feet. She had suffered from asthma in middle life. There was no history of recent ingestion of drugs.

On examination, the patient was seen to be an elderly woman with a low-grade pyrexia, which persisted during her stay in hospital. Ophthalmoscopic examination showed the blood vessels to be very narrow, and the optic discs indefinite and yellow. The retinal veins were sheathed in white and irregularly constructed. Both superficial temporal arteries were pulsatile below the zygomatic arch, but above this they were palpable as cords and were not pulsating. They were not tender. The patient had a small tender patch 2 cm. in diameter over the right parietal eminence. Her blood pressure was 160/100 mm. of mercury. The dorsalis pedis pulse was only barely perceptible in the right leg, but strong in the left. Both posterior tibial pulses could be felt. A biopsy of the temporal arteries was done by Mr. Keith Henderson, and the report (Dr. S. Clifton) was as follows:

> The changes in both temporal arteries are typical of giant-cell arteritis. They are most marked in the left vessel. In the media and especially in its deeper part there is patchy necrosis, infiltration with plasma cells and lymphocytes. Accumulation of macrophages and foreign body giant cells are seen. There is fragmentation of the internal elastic lamina. On the right side the changes appear to be of longer duration as the more acute features described above are less prominent but there is dense fibrosis throughout the vessel wall. Both vessels have a narrowed lumen. That on the left is obstructed by fairly recent thrombus which is being organized while that on the right is occluded by dense fibrous tissue traversed by narrow channels as if recanalization of thrombus has occurred. A small vein is seen with the vessel on the right but it shows none of the inflammatory changes which are present in the artery.

Investigations gave essentially normal results. The cerebro-spinal fluid was normal. The response to the Kline test was negative. X-ray examination of the skull revealed calcification in the wall of the internal carotid artery. A full blood examination revealed only mild anæmia.

The patient was treated with cortisone for two weeks without improvement, and she was discharged from hospital on January 31, 1957, with no perception of light in either eye. She was examined one month later in the out-patient department; there was no evidence of activity of the disease, but there was no perception of light in either eye.

Comment.

This disease was first described by Hutchinson (1890). He described an acute form of the disease occurring in an octogenarian who complained of red streaks on his head which were painful and prevented him from wearing his hat.

> The red streaks proved on examination to be his temporal arteries which on both sides were found to be inflamed and swollen. The streaks extended from the temporal region almost to the middle of the scalp and several branches of each artery could be distinctly traced. The condition was nearly symmetrical. During the first week when he was under my observation, pulsation could be detected in the affected vessels but it finally ceased. The tenderness then subsided and the vessels were left impervious cords.

The next reference appeared in 1930, when Schmidt described a case of temporal arteritis associated with a paracentral scotoma, and since then many cases have been described.

The pathological features are well summarized by Parsons-Smith (1952). They are those of a subacute inflammatory process commencing in the adventitia. The vasa vasorum then become involved, and many vessels are occluded; this causes areas of necrosis to appear in the media and internal elastic lamina. The inflammation spreads longitudinally, and the internal elastic lamina is gradually destroyed, though during healing new reduplicated layers are formed. The media becomes chronically inflamed, being infiltrated with lymphocytes, plasma cells and large mononuclear cells. A giant-cell reaction may be excited in the media. The intima is much thickened, and in the smaller vessels thrombosis is a common sequel. Prognosis is relatively good as regards life, though, in view of the generalized nature of the disease process, it may be fatal in some cases. Cardell and Hanley (1951) have found that in 12 of the 27 fatal cases on record, the patients have died from cerebral vascular lesions.

Various ophthalmic complications occur in the course of the disease, as would be expected, since various spots

of the ophthalmic artery are involved. Obstruction to the central retinal artery, retinal phlebitis, strabismus, ptosis and changes in the pupil may occur. Organic and nervous signs may be apparent, and mental symptoms have been recorded. Signs of involvement of other arteries, such as the femoral and brachial, occur. It appears that blindness in one eye occurs in about 27% of cases, and bilateral blindness occurs in 38%. A certain amount of vision is sometimes recovered.

The ætiology of the condition is at present unknown; consequently treatment is unsatisfactory. Cortisone appears to be of value in relieving the pain and the symptoms, and the anticoagulants, if used early, may be of value in preventing thrombosis. The present patient was seen too late for any of these measures to be of much avail, although she was treated and relieved with cortisone. However, while under cortisone therapy, she developed a peptic ulcer.

This case is reported to stress the need for prompt and energetic treatment in dealing with patients suffering from temporal arteritis if blindness is to be prevented.

Acknowledgement.

The writer wishes to thank Dr. K. O'Day, under whose care this patient was admitted to St. Vincent's Hospital, Melbourne.

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SPONTANEOUS GAS GANGRENE OF THE ABDOMINAL WALL.

By THOMAS ANTONIE, F.R.C.S. (England), F.R.A.C.S.,
St. Vincent's Hospital, Melbourne.

IN CIVIL PRACTICE gas-gangrene infection is not commonly encountered. When it does occur, it is usually in association with an open injury, particularly a compound fracture or a puncture wound of a deep penetrating nature. The infection is brought about by external contamination.

From time to time, reports are received of autoinfection by organisms occurring in the patient's own alimentary tract. Cases have been described, in which gas gangrene followed the removal of an inflamed appendix. In other instances gas-gangrene infection of the perineum has resulted from the incision and drainage of ischio-rectal abscesses. But there is invariably an external wound by means of which the specific organism has been insinuated into the tissues under anaerobic conditions. For similar considerations, the unnecessary and painful practice of attaching towel clips directly to the patient's skin is to be avoided, as the small puncture thus produced may well be the initiating point in an infection of this nature.

However, I have not previously encountered a case of gas-gangrene infection arising without any history of external wound.

Clinical Record.

The patient in question, a woman, aged 63 years, had attended the diabetic clinic for some time. For many years she had had a large epigastric hernia. It was never completely reducible, and it gave rise to much discomfort by its weight and pendulous dependency. Nevertheless, her symptoms had been largely controlled by the use of an abdominal belt. About Christmas, 1958, during a particularly hot spell of weather, she became conscious of more than the usual amount of discomfort. Her doctor manipulated the hernia in an endeavour to replace it, but without success. Subsequently, the abdominal pain persisted, and later redness developed over the most dependent part of the hernia. She endured this for some two weeks, by which time the skin had commenced to darken. It was then that she reported to the casualty department at St. Vincent's Hospital.

Examination of the patient showed her to be a grossly obese, middle-aged woman with a very large incisional hernia. The fundus of the sac hung down well below the pubes, the skin over it being extremely thinned. There was atrophy of the subcutaneous fat over the hernia, the coverings of which were affected by moist desquamation with blackenings of gangrene (Figure I). This extended over a large area, roughly circular in outline, with a diameter of some 15 in. At the flanks, particularly on the right side, this change merged into a red cellulitic zone extending posteriorly. At first the exact nature of this gangrenous process was obscure, but it was apparent that surgical drainage of the deeper tissues was necessary.

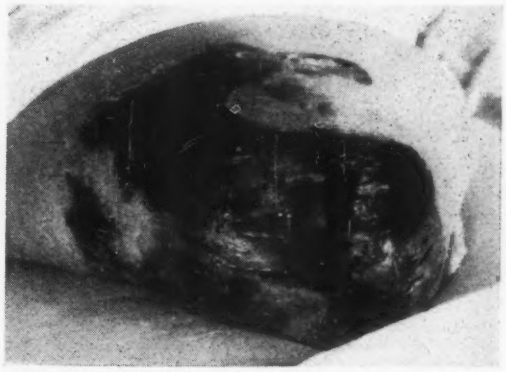

FIGURE I.

Showing the large pendulous umbilical hernia with extensive gangrene of the overlying skin. Numerous incisions are apparent exuding purulent fluid and gaseous exudate.

FIGURE II.

Showing the abdominal hernia after resolution of the infection and excision of sloughs and dead skin. The large peritoneal sac can be seen covered only by granulations.

The blackened areas were found to be quite insensitive, and accordingly multiple incisions were made without the necessity of anæsthesia. With the incision of the skin and subcutaneous tissues, evil-smelling, thin, brownish pus mixed with small masses of crepitant air bubbles exuded freely to the surface. There was no evidence of external injury, and no portal of entry for organisms could be found. But the presence of gas was suspicious

of clostridial infection, and a swab was taken for examination and culture under anaerobic conditions. Meantime the patient was given large doses of crystalline penicillin. However, her general condition continued to be reasonably satisfactory despite the massive area of gangrene and the obvious extension of the infection.

Helped by drainage, the gangrenous skin dried and separated, leaving a surviving island of skin in the centre

FIGURE III.

Showing the fundus of the hernia manually retracted vertically from the abdomen to demonstrate the hiatus in the musculature forming the relatively narrow neck.

of the hernia connected by an isthmus to the rest of the abdomen in the left flank. In due course the bacteriological report revealed the infecting organisms as *Cl. welchii* mixed with *Proteus. vulgaris*. Both were sensitive to tetracycline and the clostridia to penicillin. Once the diagnosis was established, 600,000 units of gas-gangrene antiserum were given in addition to the other antibiotics.

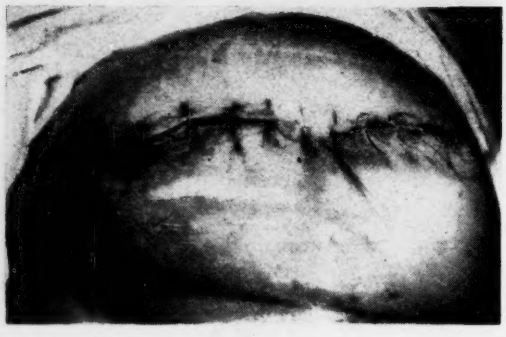

FIGURE IV.

Showing the final repair of the hernia.

Two days after her admission to hospital and the initial incisions, the patient was taken to the operating theatre with the object of opening up pockets of infection to provide freer and better drainage. Once the dead skin had been excised, the very large incisional hernia was found to be covered only by its sac of peritoneum surrounded by a layer of granulation. Even under anæsthesia the contents of this sac could not be reduced. However, the neck was seen to be relatively narrow by comparison with the fundus, and disappeared through a hiatus in the abdominal wall measuring some 5 in. by 4 in. With the dependent sac elevated, a large amount of necrotic cellulitic tissue, consisting largely of abdominal fat, was exposed. This tissue, which extended into either flank, was excised widely till free bleeding and healthy tissue were encountered. After all pockets had been opened to the air, drain tubes were insterted and the wound was lightly covered. In addition to antibiotic therapy, potassium permangate and hydrogen peroxide irrigations were carried out several times a day. Under this régime the infection rapidly subsided. Nevertheless, to judge by repeated bacteriological examinations, it was ten days before all clostridia were completely eradicated. Ultimately the wound healed, leaving a very large area of open granulating surface across the whole of the abdomen with the large hernial sac contained in the centre (Figures II and III).

Subsequently, the patient was again taken to the operating theatre. The sac was opened, and was found everywhere to be adherent to its contents, which consisted of intestine, both small and large, and a considerable amount of the omentum. After tedious dissection it was freed, and with coaxing and manœuvring the abdominal contents were once again reduced within the peritoneal cavity proper. The deficit left in the abdominal wall was fairly easily repaired according to the method of Mayo. As the patient was obese, there was redundancy of the abdominal skin, which allowed loose closure to be effected after drain tubes had been inserted (Figure IV). The patient progressed well from this operation, and a complete convalescence seemed assured. Unfortunately on the eighth day she suffered a sudden collapse, apparently from massive pulmonary embolism, and died in a state of sustained shock.

Comment.

I report this case because I have not previously encountered, or heard of, gas-gangrene arising spontaneously in the abdominal wall, or indeed elsewhere, without evidence of some external wound.

One assumes in this case that the irreducible contents became inflamed and irritated, permitting transmigration of infecting organisms from within the lumen of the intestine to the outer coverings of the sac. Apparently *Cl. welchii* were natural inhabitants of the intestinal tract. When free to enjoy anaerobic conditions in the layers of the abdominal wall, they rapidly flourished to produce the necrotizing inflammation associated with gas development.

MANIA DUE TO CEREBRAL FAT EMBOLISM FOLLOWING FRACTURE OF THE TIBIA.

By H. S. SYMONS,
Senior Resident Medical Officer, Psychiatric Ward, Royal Melbourne Hospital, Melbourne.

Clinical Record.

THE patient was a Sicilian woman, aged 28 years, who sustained a transverse compound fracture of the left tibia on February 23, 1959. There was a skin laceration 0·25 in. long over the subcutaneous border of the tibia. A closed reduction was performed at the Dandenong Hospital under general anæsthesia and a full leg plaster splint applied. Tetanus antiserum (1500 units) was administered. There was no history available of abnormal behaviour prior to the accident.

Four days after the accident the patient became noisy, non-cooperative and difficult to manage. She was admitted to the Royal Melbourne Hospital on March 2, 1959, under the care of Dr. A. J. M. Sinclair and Mr. B. T. Keon-Cohen. On examination, the patient was garrulous and exceedingly voluble, and her speech was elaborated by frequent gestures. However, the patient's communication in English was not good. Contact with reality was good. A pulse rate of 120 per minute and a temperature of 37·5° C. were the only abnormal physical findings.

She was sedated with chlorpromazine, 100 mg. every four hours, pentobarbitone, 400 mg., as the necessity

arose, and paraldehyde, 10 ml. every four hours when required. Despite such heavy sedation she remained noisy, and a course of electroconvulsive therapy was commenced on March 4; 30 such treatments were given over the next 14 days.

The chronological relationship between the onset of a manic illness and the occurrence of the fracture suggested the possibility of fat embolism. A microscopic examination of the urine revealed numbers of fat globules. No abnormal neurological signs were detected. X-ray films of the chest and skull revealed no abnormality. The lumbar cerebrospinal fluid presented no abnormality, and the Wassermann test produced a negative result. The sputum was yellowish and mucoid, but was not examined microscopically.

The patient continued to have a low, intermittent fever, and persistent tachycardia was present. Penicillin was given as a prophylactic measure in a dosage of 600,000 units every six hours. A state of excitement resembling that of cerebral irritation persisted for two and a half weeks and indeed was so severe at times that 0·25 to 0·5 gramme of amylobarbitone given intravenously was required. At the end of the first week of treatment, the patient was amnesic, disorientated and incontinent of urine and fæces, and the original features of the abnormal mental state remained unchanged. Three to four litres of fluid with a high protein content were given daily through a Ryle's tube in order to compensate for loss of fluid by sweating.

Heparin therapy was begun on March 6, 40,000 units being given intramuscularly every day in four divided doses. Heparin was employed because of its action in fat mobilization and dispersal of chylomicrons in the blood. Whilst it was recognized that this would have little effect on already occluded vessels, it was considered it might have some action in preventing the occurrence of further emboli.

Carbon dioxide (5%) in oxygen was administered on March 19 via a face mask at the rate of 5 litres per minute for two hours, in order to ascertain whether there were any favourable effects as a vasodilator.

On March 20 the patient was much quieter and more cooperative, and was able for the first time to discuss her condition. It now became possible to relax restraint. Left basal bronchopneumonia, which was present, resolved after the exhibition of penicillin, streptomycin and tetracycline.

The patient's condition continued to improve, but eleven days later relapsed, when she again became voluble, pseudo-religious and antagonistic towards her husband. Discussion through an interpreter revealed this to be a reaction to a disagreement with her husband. She continued to improve with reassurance, and remained a cheerful, cooperative and placid person.

After a change of plaster cast on April 12, physiotherapy was undertaken, and the patient quickly learned to manipulate crutches and was discharged home on April 15.

Differential Diagnosis and Discussion.

The main differential diagnosis was between acute mania and cerebral fat embolism. Aspects in favour of the latter included the significant time relationship of the fracture to the onset of cerebral symptoms, the presence of chyluria and the failure of electro-shock therapy to produce remission. It would be expected that acute mania would respond to 30 electro-shock treatments administered over such a short period of time. Further, the absence of a history of previous attacks of mania is significant. Cerebral fat embolism is most often accompanied by delirium or a state of cerebral irritation. H. W. Pia (1957) states:

> If, following a fracture or severe contusion of the soft parts, without cranial or cerebral involvement, there is found, after a free interval, possibly with pulmonary symptoms, a dulling of consciousness or a delirium pattern, there can hardly be any doubt about a diagnosis of cerebral fat embolism.

In view of the hysterical outburst after her initial improvement, the possibility that the entire episode was purely hysterical must be considered. However, it would be unusual for an hysterical illness to remain unchanged after such a régime of electro-shock therapy.

This case suggests that a well-defined psychotic condition such as mania may be released by an organic cerebral illness such as cerebral fat embolism. It may be speculated that the mania was "fired off" by a focal cerebral disturbance resulting from alterations in blood supply, by abnormal electrical discharge, or by biochemical, ionic or pH variation.

The use of electro-convulsive therapy in this case is obviously open to question; but it did appear to be of some value as a method of controlling excitement. The main factors in ultimate recovery appear to have been good nursing care, sedation, chemotherapy for infective complications and the passage of time. The fact that the first sign of improvement occurred shortly after "Carbogen" inhalation is of doubtful significance.

The only recent monograph available on the problem of cerebral fat embolism is that of H. W. Pia, of Giessen University, Germany (1957). On the basis of systematic post-mortem examinations, he suggests that there is a higher incidence of cerebral fat embolism than is at present recognized. The pathological changes described include annular hæmorrhages in the medulla and miliary foci of softening (moth-eaten foci) to be found in the cortex. Pia reports five cases; all the patients survived, and all had disturbances of consciousness for one, two, four, five and 13 weeks respectively. Treatment was in the main symptomatic, but an intravenous infusion of "Novocain" was used in all cases.

Summary.

The case described is that of a Sicilian woman, aged 28 years, who sustained a fracture of the tibia, followed four days later by signs of cerebral irritation and manic behaviour. The finding of chyluria suggested that this state was due to cerebral fat embolism. Recovery occurred after two and a half weeks. Treatment consisted of electroconvulsive therapy, heavy sedation, chemotherapy and careful nursing care. Heparin and "Carbogen" were also used for short periods of time. The case is of interest in that mania could be released by an organic illness such as cerebral fat embolism.

Acknowledgements.

My thanks are due to Dr. A. J. M. Sinclair, Honorary Psychiatrist to the Royal Melbourne Hospital, and Mr. B. T. Keon-Cohen, Honorary Orthopædic Surgeon to Royal Melbourne Hospital, for permission to publish details of this case.

Reference.

PIA, H. W. (1957), "Differential Diagnosis and Treatment of Cerebral Fat Embolism", *Arch. klin. Chir.*, 287: 677.

Reviews.

Clinical Orthopædics, 13. Anthony F. DePalma, editor-in-chief, with the assistance of the associate editors, the Board of Advisory Editors and the Board of Corresponding Editors; Number Thirteen, Spring, 1959. Philadelphia and Montreal: J. B. Lippincott Company, Limited. Sydney: Angus & Robertson, Limited. 10″ × 6¾″, pp. 406, with many illustrations. Price: £3 6s.

THIS book is concerned mainly with the surgery of the hand. There are 18 separate papers in the first section dealing with various aspects of hand surgery. These comprise about two-thirds of the whole volume. There is another paper on Dupuytren's contracture placed amongst miscellaneous items later on. Most of the articles on hand conditions are written by surgeons with large experience in this speciality, and their work and reputation are well recognized. Together they give a very well-ordered and comprehensive review of this subject.

The second and third sections of the volume deal with a variety of orthopædic conditions, including a paper on the

role of the disks of the sterno-clavicular and acromio-clavicular joints by the editor-in-chief, Anthony De Palma. There is probably something to interest everybody amongst the odd and interesting subjects which are discussed in this final third of the volume. The contributions, however, do not maintain the same standard as those dealing with the hand, and they detract from rather than enhance the value of this particular volume.

This is the thirteenth volume of this series of "Clinical Orthopædics", and we are promised further numbers of the same size up to No. 21, which is due in America in the fall (Australian spring) of 1961.

Open Reduction of Common Fractures. By Oscar P. Hampton, Jr., M.D., F.A.C.S., and William T. Fitts, Jr., M.D., F.A.C.S.; 1959. *Modern Surgical Monographs*, editor-in-chief, I. S. Ravdin, M.D.; consulting editor, Richard H. Orr, M.D. New York and London: Grune & Stratton, Inc. 9" x 6", pp. 224, with many illustrations. Price: $8.75.

THIS small volume is true to title, in that it provides a concise account of the treatment of those fractures which are unsuitable for more conservative measures. The authors appreciate that the open reduction and the metallic fixation of fractures are calculated risks affording several advantages but carrying some definite disadvantages. They believe that the majority of the latter can usually be circumvented by proper selection of patients for this method of management and by avoiding technical pitfalls. No appeal is made in the text for the routine use of open reduction as a method of choice; the book can therefore be considered complementary to the better-known "Closed Treatment of Common Fractures" by John Charnley.

The illustrations and line drawings are clear; but some reproductions of the X-ray films are of poor quality and many have been retouched. Sufficient details of technique are given to make this volume useful to the occasional orthopædic surgeon, and the description of each fracture includes a "Pitfalls and Precautions" summary, which is excellent. The book can be recommended as a worthwhile reference when the larger texts are not available.

The Child with Abdominal Pains. By John Apley, M.D., F.R.C.P.; 1959. Oxford: Blackwell Scientific Publications. 8¾" x 5½", pp. 96. Price: 12s. 6d. (English).

IN his preface, the author states that he resisted the temptation to call this monograph "Little Belly-Achers", partly because it carries a suggestion of irritation on the doctor's part, an irritation likely to be increased by the many gaps in our knowledge. He then sets out to fill in some of the gaps.

The first half of the book is a detailed study of 100 consecutive children admitted to hospital with recurrent abdominal pain, and of another 108 children with similar symptoms found by routine questioning of 1000 children at school medical examinations. The study included the parents and the entire domestic background. There follows an excellent general discussion on the causation of recurrent abdominal pain, in the course of which the author builds up his thesis that, in the very large majority of cases, no primary underlying organic basis exists. Some of his aphorisms are worth quoting: "Pain is an experience in which the whole family takes part". "Anxiety, like courage, is contagious", "The farther the localization of the pain from the umbilicus the more likely is there to be an underlying organic disorder".

In the diagnosis of vague diseases, we all tend to find what we habitually look for. Whether this is so in the present case is for the reader to decide; but at least the facts are fairly presented and the conclusions are sound. Whether the author has a bias towards emotional disorders only his colleagues know; but he quotes a letter from one of them who, in referring a patient, wrote: "Kindly examine the soma and leave the psyche to me."

Clinical Dermatology: For Students and Practitioners. By Harry M. Robinson, Jr., B.S., M.D., and Raymond C. V. Robinson, B.S., M.D., M.Sc. (Med.); 1959. Baltimore: The Williams & Wilkins Company. Sydney: Angus & Robertson, Limited. 10½" x 7¼", pp. 258, with 116 illustrations. Price: 93s. 6d.

THIS concise and compact book of just over 200 pages must be considered one of the most useful aids in the teaching of diseases of the skin which has been produced in recent years.

The work is of an essentially practical nature, and the authors and collaborators have most ingeniously contrived to include both common and rare skin diseases in a very small space. This has been achieved by a succinct standardized method of description of each disease, and by the elimination of long and tedious passages in adjectival skill, the place of which is taken by numerous monochrome clinical photographs of high-class quality. The authors thus realize the great value of visual aids in the teaching of dermatology. If any criticism were offered, it would be to the effect that these illustrations were not in colour; but it must be remembered that to reproduce so many excellent black-and-white illustrations in colour of equally good quality would add to the difficulties and cost of production.

The book is divided into two main sections. Part I deals with "General Procedures"—histopathology, mycology, allergy, therapy, etc.—while Part II, "Morphologic Dermatology", includes regional involvement, charts of differential diagnosis, morphological diagnosis, etc. In this section, incidentally, is an excellent specimen of the practical nature of the book, exemplified by the heading "Eruptions which Rarely Involve the Face".

A praiseworthy restraint is exercised in avoiding systematized classifications, so much a feature of the majority of large textbooks. Emphasis is on clinical classification, and there is a minimal effort to place diseases in strict "watertight compartments".

The general setting-out of the book, ease in reading, format and quality of paper leave little to be desired. The work is highly recommended for student and practitioner alike. For the specialist it is a handy volume for speedy reference, but, above all, a most valuable aid in teaching.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Recent Research in Freezing and Drying", edited by A. S. Parkes, C.B.E., F.R.S., and Audrey U. Smith, D.Sc., M.B., B.S.; 1960. Oxford: Blackwell Scientific Publications. 9¾" x 6", pp. 328, with many illustrations. Price: 63s (English).

"Current Approaches to Psychoanalysis", edited by Paul H. Hock, M.D., and Joseph Zubin, Ph.D.; 1960. New York and London: Grune & Stratton, Inc. 8½" x 5½", pp. 226 Price: $6.50.

"Babies and Young Children: Feeding: Management Care", by Ronald Illingworth and Cynthia Illingworth. Second edition; 1960. London: J. & A. Churchill, Limited 8½" x 5½", pp. 339, with 23 plates and many illustrations Price: 18s. (English).

"Modern Nutrition in Health and Disease: Dietotherapy", edited by Michael G. Wohl, M.D., and Robert S. Goodhart, M.D., with 59 Contributors; Second edition; 1960. Philadelphia: Lea & Febiger, Limited. Sydney: Angus & Robertson, Limited. 9¼" x 6", pp. 1152, with 75 illustrations and 154 tables. Price: £10 3s. 6d.

"A Handbook of Diseases of the Skin", by Herbert O. Mackey, F.R.C.S.I., L.R.C.P.I., D.P.H. (Univ. Dub.), L.M., F.R.I.A.M.; Sixth edition; 1959. London: Macmillan and Company, Limited. Dublin: C. J. Fallon, Limited. 8¾" x 5¼", pp. 264, with 203 illustrations. Price: 8s. 6d. (English).

"Defective Molecules as a Cause of Disease: An Inaugural Lecture", by G. H. Lathe, M.Sc., M.D., C.M., Ph.D.; 1960. Leeds: Leeds University Press. 8½" x 5½", pp. 30. Price: 2s. 6d. (English).

"Pharmacology and Therapeutics: A Textbook for Students and Practitioners of Medicine and its Allied Professions", by Arthur Grollman, Ph.D., M.D., F.A.C.P.; Fourth edition; 1960. Philadelphia: Lea & Febiger, Limited. Sydney: Angus & Robertson, Limited. 9¼" x 6", pp. 1080, with 217 illustrations. Price: £6 17s. 6d.

"Surgery and Clinical Pathology in the Tropics", by Charles Bowesman, O.B.E., B.A., M.D., F.R.C.S.E., F.A.C.S., D.T.M. & H.Ed.; 1960. Edinburgh and London: E. & S. Livingstone, Limited. 9½" x 6", pp. 1076, with 321 illustrations. Price: £5 10s. (English).

"Peptic Ulceration: A Symposium for Surgeons", by Charles Wells and James Kyle, M.B., M.Ch., F.R.C.S., F.R.C.S.(I.), with a foreword by Lester R. Dragstedt; 1960 Edinburgh and London: E. & S. Livingstone, Limited. 9¾" x 6½", pp. 272, with 44 illustrations. Price: 42s (English).

The Medical Journal of Australia

SATURDAY, JUNE 18, 1960.

THE TREATMENT OF TRACHOMA.

TRACHOMA is one of the major scourges of man, particularly in tropical and sub-tropical regions. It is characteristically prevalent in places where most of the people are very poor and underprivileged, and this in itself creates extra problems and in many ways directs the pattern of its management. In particular, it determines that the cost of treatment is of great importance. It is in just such situations as this that the role of the World Health Organization is best seen, both in the advice that it is able to offer from its resources of experience and in the practical aid it can provide from its resources of finance and trained personnel. Its programme for any country with endemic trachoma is in five parts: first, a preliminary survey to study the nature and extent of the problem and the mode of spread of the disease; second, pilot projects in which different forms of treatment are tried out; third, a mass campaign based on the results of the pilot projects; fourth, whenever possible, laboratory investigation of the virus; fifth, adoption of treatment and prophylaxis of trachoma as part of the routine procedure of the public health department concerned. In this way it has been possible for patterns of treatment and control to be worked out and applied in particular countries, and at the same time for common resources to be called upon and the experience gained from work in one country to be added to the common knowledge. An important experience in this respect is that recounted by J. Reinhards, A. Weber and F. Maxwell-Lyons[1], who report on the collective antibiotic treatment of trachoma in Morocco, and in particular on comparative trials leading to more economical methods of treatment.

It has been known from the early 1950's that certain wide-spectrum antibiotics are effective against the causative agents of trachoma. It seemed, however, that treatment had to be continued over long periods to effect a cure in the average case. This was expensive, especially where thousands of patients had to be treated, and so was impracticable in places where trachoma was rife and poverty was general. In the Morocco study comparative trials were carried out with the assistance of W.H.O. on an unprecedented scale, involving more than 9000 school children with active trachoma. The object was to assess the relative values of different methods of local treatment of trachoma with chlortetracycline. The trials were carried out on school children in selected schools in Morocco, for it was considered impossible to keep all the children in two towns under close observation (approximately 12,000 in Marrakech and 7000 in Meknès). The form of treatment used was the application of a 1% ointment of chlortetracycline direct to the eyelids. In 1954-1955 a group of 3828 trachomatous children were treated three times daily for five days of the week for 60 days, the schools being closed for two days a week. In 1955-1956, on three separate groups totalling 4136 trachomatous children, three different schedules of treatment were studied. The first schedule was as above; in the second schedule the ointment was applied twice a day for 60 consecutive working days; in the third schedule the ointment was applied twice daily on three consecutive days every four weeks over a period of 20 weeks—i.e., there were six three-day cycles of treatment. In 1957-1958, two groups totalling 11,067 children in a rural area were treated to assess the relative value of the first and third schedules under rural conditions. It was recognized that individual subjects of trachoma varied in their susceptibility to antibiotics, and that in any large-scale programme of collective treatment cures could not be obtained in all cases. Taking the practical view of the problem, the medical team agreed to work to an "acceptable limit of failure", on the grounds that it is more economical to re-treat a slightly larger number of patients for relapses than to subject all patients to an unnecessarily generous course of treatment in the first instance.

At the end of the first series in 1954-1955, it was found that in 56·9% of active cases of trachoma, the patients were clinically cured, the greater number of cures being in the early cases. In all the failures the patients were re-treated. Triple sulphonamides were then given in addition to chlortetracycline ointment, and at the end of the treatment 77·9% of the treated patients were clinically cured. In the 1955-1956 series the cure rates from all three schedules were very much the same: from the first schedule 69·6%, from the second schedule 69·6% and from the third schedule 71·1%. Comparable results were obtained from all three schedules, whatever the stage and severity of the trachoma. In all cases younger children reacted less satisfactorily than older children. In the 1957-1958 series, in which the children were from rural surroundings, secondary infections were more prevalent and the general living conditions on a lower level than in the northern towns. No difference could be found between the respective cure and failure rates in the two groups.

It is clear that trachoma in Morocco is susceptible to treatment with antibiotics. Treatment with chlortetracycline has resulted in clinical cure in 57% to 80% of cases of trachoma in Moroccan school children. With re-treatment of the failures more than 98% of the school children with trachoma were cured. A consideration of the results from the three different schedules of treatment shows that the local application of 1% chlortetracycline ointment three times a day over 60 consecutive working days has resulted in about 80% of cures in the better-class urban schools, that under similar conditions the frequency of application can be reduced to twice a day without loss of efficacy, and that equally good results follow short-term

[1] *Bull. Wld Hlth Org.*, 1959, 21: 665.

treatment over long periods—for example, application of chlortetracycline twice a day on three consecutive days at intervals over a period of twenty weeks. The intermittent treatment shows many advantages over the 60-day treatment. The consumption of ointment for a full course of treatment for one person is reduced from 18 grammes to between 3 and 6 grammes. One instillator is able to treat three to five times as many children by intermittent methods. Intermittent treatment interferes less with school work. The importance of the economic consideration can be seen from the number of children treated and re-treated. In 1958 altogether 263,000 children were treated in Morocco. The target number for 1959 was 350,000, and that for 1960 is 400,000. Reinhards, Weber and Maxwell-Lyons state that minimum requisite treatment with antibiotics may vary from country to country, and that local conditions may make it advisable to have different intervals between the cycles of treatment. Other antibiotics than chlortetracycline may be useful—for example, they cite results of intermittent treatment in a Spanish village, where oxytetracycline was used and the cure rate was 96·3%. There is also a report of 70% of cures in South Africa after intermittent treatment with chloramphenicol.

It should be noted that the emphasis in this report is on treatment with locally applied antibiotics. The value of the sulphonamides was recognized, but their use was regarded as an auxiliary measure and not adopted as a routine. Other workers, e.g., F. Flynn in the Northern Territory,[2] have preferred to adhere to the W.H.O. recommendation of combined treatment with sulphonamides given by mouth and antibiotics applied locally, and their results have been satisfactory. In Western Australia, in the campaign being carried on by the Department of Public Health (to which we shall return on another occasion), the antibiotics have been discarded because of practical difficulties, and the sulphonamides alone have been relied upon—again with satisfactory results. Reinhards, Weber and Maxwell-Lyons point out that suitable treatment of trachoma will vary from country to country in accordance with the characteristics of the local disease, and well-controlled pilot trials are necessary in each case. They abandoned the use of sulphonamides in Morocco because of "the high cost and potential dangers of mass treatment" with them, and because antibiotics used alone had proved sufficient. Others have had quite a different experience and have been satisfied with a carefully chosen sulphonamide. In any case it seems that, despite varying circumstances, successful measures can be devised for mass treatment of trachoma at what will be regarded locally as an economically acceptable level.

[2] MED. J. AUST., 1957, 2:269 (August 24).

Current Comment.

CHLORPROMAZINE AND THE OUT-PATIENT.

THE use of chlorpromazine in the treatment of acute psychotic illnesses characterized by restlessness, excitement and agitation is regarded by many people as one of the major advances in psychiatric treatment in the last fifty years, and papers too numerous to mention have testified to its value. Its use in chronic hospitalized psychotics has been equally spectacular although not so fully documented. Its arrival on the therapeutic scene facilitated attempts to open the doors of chronic wards and to apply to them the principles of the therapeutic community, so that our asylums have been to a large extent transformed into psychiatric hospitals in the true sense of the term. This transformation is so complete that a new generation of psychiatrists is emerging which has never experienced the frustration of trying to cope with disturbed back wards containing rows of restless, shouting psychotics. Even hospital architecture has been transformed, so that single rooms are no longer places for the confinement of violent patients, but are designed as individual bedrooms providing the tranquillized patient with the comfort and privacy to which he is accustomed outside hospital.

The use of chlorpromazine in the treatment of psychotics on an out-patient basis is not so well documented as its use with in-patients. All psychiatrists must have had the satisfaction of preventing the admission to hospital of early psychotics by the timely use of chlorpromazine on an out-patient basis. All too must have seen the rapid relapse and return to hospital of ex-patients of mental hospitals who have omitted for one reason or another to continue taking the maintenance dose of chlorpromazine on which they were discharged. Such clinical impressions are difficult to evaluate scientifically, and hence the paucity of contributions to the literature on this aspect of the use of chlorpromazine. However, although few, a number of reports do exist which lend statistical support to these impressions.

In 1957 N. W. Winkelman[1] reported that, as the result of using chlorpromazine with out-patients, 40% of patients who would formerly have been hospitalized were able to be treated at home or in the community. He emphasized the high relapse rate after withdrawal of chlorpromazine, and stated that maintenance therapy must be continued indefinitely unless the basic pathology was altered. H. C. B. Denber and E. G. Bird,[2] who reported a study of 1523 patients treated with chlorpromazine, emphasized that convalescent patients must be followed closely after leaving hospital, as relapse might occur within a short time if the maintenance dose was not regulated accurately. They stated further: "Our observations indicate that patients with repeated psychotic attacks should be on a maintenance dose of chlorpromazine for extended periods." F. Labhardt[3] reported a follow-up study of the chlorpromazine treatment of 373 schizophrenics. He found that most relapses occurred, irrespective of whether the illness was of recent onset or of medium or long duration, when the patient was receiving either no maintenance medication or a dose which was too low. He recommended that in recent cases of schizophrenia treatment should be continued with chlorpromazine for 6 to 8 months, and that in subchronic and chronic cases treatment should be long-continued with more or less large maintenance doses. E. B. Kris and D. M. Carmichael[4] observed a series of female patients who had had previous psychotic episodes—a group which is usually recognized as being particularly prone to recurrence of the psychosis during the stress of pregnancy and the puerperium. They found that the administration of chlorpromazine, during pregnancy and the puerperium, to these patients seemed to be of definite value in preventing a recurrence of the psychosis.

In 1959 P. N. Wold,[5] in an admittedly small series of 6 patients with chronic schizophrenia who had been out of hospital for a year, found that a major relapse occurred in 5 of the 6 patients when a placebo was substituted for the chlorpromazine. They improved when chlorpromazine therapy was reinstated, and relapsed for a second time when it was again replaced by the placebo. W. Tuteur, R. Stiller and J. Glotzer[6] reported the use of chlorpromazine in chronic hospitalized schizophrenics

[1] *Amer. J. Psychiat.*, 1957, 113:991.
[2] *Amer. J. Psychiat.*, 1957, 113:972.
[3] *Schweiz. Arch. Neurol. Psychiat.*, 1957, 80:275.
[4] *Psychiat. Quart.*, 1957, 31:690.
[5] *Amer. J. Psychiat.*, 1959, 116:[illegible].
[6] *Illinois med. J.*, 1959, 116:9.

They found that 258 out of 822 patients were enabled by its use to leave hospital, thus considerably reducing the overcrowding in their chronic wards. These patients were followed up as out-patients, and 100 have been out of hospital for over 2 years. A placebo trial was carried out on a number of patients who had been out of hospital for some months. This resulted in relapse in 59·7% of cases, with symptoms similar to the pre-treatment picture. Most of the relapsed patients improved within several days of receiving chlorpromazine again. Tuteur, Stiller and Glotzer believe that their findings indicated that chronic schizophrenics should continue to take chlorpromazine for years after discharge from hospital.

There is thus very substantial support for the belief that chlorpromazine administered at the out-patient level can and does keep patients out of hospital, to the benefit of themselves, their families and their status in the community. The only drawback to its use is its high cost. This disadvantage is met only partly by the supply of chlorpromazine by out-patient clinics and mental hospitals to discharged patients, and the cost factor deters many patients who would otherwise do so from seeking treatment from the psychiatrist of their choice in private practice. Many patients by reason of the high cost of the drug stop taking it too soon, and on their subsequent relapse have to be returned to hospital. An excellent case can be made out for the inclusion of chlorpromazine in the Pharmaceutical Benefits formulary for specified patients, as the cost to the Government of making it available to an out-patient is far less than the cost of maintaining the same patient in a mental hospital. It is not suggested that it be made generally available, but its supply as a pharmaceutical benefit restricted to those who really need it should not present an insuperable administrative problem.

DIHYDROSTREPTOMYCIN DEAFNESS.

WE are once again reminded of the dangers which attend the prescription of many potent modern drugs by a report, by G. E. Shambaugh and seven of his colleagues,[1] which draws attention to the particularly insidious dangers of dihydrostreptomycin. These authors state that attention was drawn in 1954 to the permanent deafness which might occur after comparatively small doses of dihydrostreptomycin, but that cases of irreversible hearing loss attributable to this antibiotic are continuing to occur. This is often without the knowledge of the prescribing physician because of the latent period, which may be as long as six months between the administration of the drug and the onset of hearing loss. The authors point out that this latent period is unique for dihydrostreptomycin; with other drugs which cause deafness, such as quinine, streptomycin, neomycin and kanamycin, loss of hearing usually occurs immediately or very soon after administration. Both streptomycin and dihydrostreptomycin have a toxic effect on the hearing mechanism, but streptomycin acts mainly on the vestibular function and dihydrostreptomycin on the cochlear function. It was hoped that by combining the two in equal parts, the total dosage of each would be reduced and the risk of damage to hearing thereby lessened. However, the authors point out that what was first thought to be a logical therapeutic improvement has in fact turned out to be an added danger, especially in the case of certain proprietary preparations which contain dihydrostreptomycin combined with penicillin and streptomycin, under names which do not clearly indicate the presence of dihydrostreptomycin. In a series of 32 patients who suffered hearing loss as a result of medication with dihydrostreptomycin there were several who had been given such combined preparations prophylactically after a surgical operation, or for some quite trivial complaint. We do not know whether any such combined preparations are marketed in Australia, but such cases are an excellent illustration of the dangers of "shotgun" therapy. Dihydrostreptomycin must be regarded as an unusually treacherous drug on three counts: the long latent period which may elapse between the administration of the drug and the onset of hearing loss, the fact that serious irreversible hearing loss may occur after doses totalling as little as two or three grammes, and the fact that the prescribing doctor may never know the sequel to his prescription. It is therefore not surprising that Shambaugh and his colleagues recommend that dihydrostreptomycin should not be included in combined antibiotic preparations unless this is clearly indicated in the name of the preparation. They further conclude that since streptomycin is as effective as dihydrostreptomycin against Gram-negative and acid-fast bacteria, and since toxic reactions due to streptomycin occur immediately, are more easily recognized and are less permanently disabling, there seems to be little reason to use the more dangerous drug. This report was sponsored by the Committee on Conservation of Hearing of the American Academy of Ophthalmology and Otolaryngology.

[1] *J. Amer. med. Ass.*, 1959, 170: 1657 (August 1).

BIOPHYSICAL SCIENCE.

IN July-August, 1958, the Biophysics and Biophysical Chemistry Study Section of the United States National Institutes of Health held a meeting in Boulder, Colorado, to which about 120 senior research workers and younger scientists were invited. The aim of the meeting was to promote the bridging of the gap between the concepts and methods of the physiological sciences and those of the biological sciences in the investigation of living material. The core of the study programme was a series of about 60 lectures constituting compact summaries of certain key problems and critical evaluations of recent advances. The papers were mainly directed towards those whose basic training was in the fields of physics or physical chemistry. For the most part they were concerned with molecular biology, which is the term now being widely used for the study of the physical-chemistry of living processes at the sub-cellular level. Thus they dealt with such problems as the ultra-structure of cells, biological macromolecules and the forces between them, protein structure and enzyme action, and the fibrous proteins of muscle. The fact that particular emphasis centred upon nerve and muscle is not surprising, since these fields have always attracted those primarily trained as physicists and chemists. A few of the papers were concerned with the interaction between cells, particularly nerve cells, and the mechanisms of impulse initiation and synaptic transmission. Current interest in information theory and communication engineering was reflected in papers on the coding aspects of protein synthesis and genetic coding generally, biological transducers and coding, and pattern recognition by the central nervous system.

Taking the papers as a whole, one cannot but be impressed by the advances made by the electron microscopist. They will probably prove to be as significant as those made by the light microscopist in the second half of the last century. All the papers presented at the meeting were published in the January and April, 1959, issues of the journal *Reviews of Modern Physics*, and at the same time they were put together in the form of a book.[1] This presumably accounts for its prompt publication, and for the fact that it is remarkably cheap for such a large and relatively specialized volume which includes many illustrations and half-tone reproductions. It has both an author and subject index covering all the papers in the volume. Although primarily directed at the physicist, this book will probably be read with equal or even greater profit by the biologist trying to understand the physical basis of his science.

[1] "Biophysical Science — A Study Program", planned and edited by J. L. Oncley, editor-in-chief; F. O. Schmitt, R. C. Williams, M. D. Rosenberg and R. H. Bolt; 1959. New York: John Wiley & Sons, Inc.; 10¼" × 7¼", pp. 138, with many illustrations and tables. Price: $6.50.

Abstracts from Medical Literature.

UROLOGY.

Physiological Response to the Ileal Bladder.

J. R. JUDE, A. H. HARRIS AND R. R. SMITH (*Surg. Gynec. Obstet.*, August, 1959) report the results of a study of the physiological response of the body in 22 patients with ileal conduit bladders, who were under observation for periods of from six months to over two years after their operation. In 14 of these patients the ileal bladder was constructed as a part of pelvic exenteration for advanced carcinoma of the cervix. Seven other patients had had treatment for carcinoma of the bladder, and in one a temporary ileal bladder was constructed to allow closure of a large uretero-vesico-vaginal fistula. The length of the ileal segment in these cases was 20 to 25 cm. A non-obstructing ileostomy opening was made in the right lower quadrant of the abdomen, and a free flow of urine into a disposable plastic bag adherent to the skin was at all times permitted. Serial studies were performed to evaluate the effect of this method of urinary diversion on the physiological mechanisms of the body. These studies revealed that during the period of observation such mechanisms were not adversely affected. Renal function was maintained or improved, blood electrolytes were not abnormally altered, and the upper part of the urinary tract was not damaged by ascending infection. The authors conclude that the ileal bladder has proved to be a valuable method of temporary or permanent urinary diversion.

Surgical Treatment of Hydronephrosis.

T. E. GIBSON (*West J. Surg.*, July-August, 1959) states that, despite continuous progress in the treatment of obstructions at the uretero-pelvic junction, there are still controversial aspects of the surgical treatment of hydronephrosis. Experience has taught that, to obtain the best results and reduce the proportion of failures, Foley's postulates must be observed. These are: (*a*) there must be no shortening of the suture line; (*b*) high insertion of the ureter must be corrected; (*c*) there must be gradual funnelling of the pelvis into the ureter. Foley's procedure, based on the original pyeloplastic method introduced by Schwyzer in 1923, is a Y-plasty with flap and sutures. Several modifications have been described in recent years, and are all relatively satisfactory. There are two types of modern procedure which possess great value in certain conditions. The Davis operation of longitudinal incision of the obstructed area with prolonged intubation and no sutures at all has introduced a fresh principle which has been widely used with success. The Nesbitt procedure, in which the ureter is first cut entirely away from the pelvis, incorporates the principle of elliptical anastomosis and introduces a safeguard of value in this type of surgery. A broad, spatulate type of anastomosis between pelvis and ureter reduces the chances of circumferential structure. The author himself now prefers a modification of the Foley procedure, and inclines towards the modern tendency of dispensing with a ureteric splint, because convalescence is shorter and more comfortable and there is less morbidity. Some surgeons also dispense with a nephrostomy drain for deviation of the urine; instead, they simply rely on a slit or vent in the wall of the renal pelvis. This allows easy escape of urine towards an extra-renal drain, thereby preventing intra-pelvic tension until the urine flows freely down the ureter. The Davis intubated ureterotomy procedure has certain definite indications, and when these are present it is a most valuable procedure. Such indications are: (i) previous operative attempts with subsequent scarring; (ii) long or multiple strictures of the ureter; and (iii) older patients, who tend to develop lasting fistulæ unless long splinting is used. In the author's modification of the Foley procedure a large area of the pelvic wall is excised. To leave a large flabby pelvis with an atonic wall is to risk a poor functional result.

Partial Nephrectomy.

F. S. HAMM AND P. FINKELSTEIN (*J. Urol.* (*Baltimore*), December, 1959) report their experiences with partial nephrectomy in 46 cases. The majority of these concerned patients with calculous disease (39); a few were cases of disease of one portion of a duplicated calyceal system (six), two of them associated with calculi; and two were cases of tuberculosis, one of these being also included in the calculous disease group. The authors conclude that this operation, properly done, is a safe and effective method of conserving renal tissue. The points of technique stressed are as follows: (i) exposure must be adequate, so that the renal vessels are fully exposed and can be lightly dissected out to study their distribution; (ii) when indicated, the uretero-pelvic junction is exposed; (iii) a nephrostomy tube is not used, as it may lead to infection and hæmorrhage; (iv) the entire diseased portion is removed and a careful closure of the calyceal wound is made; (v) a vent is incised in the renal pelvis, and a drain is left down to this point; (vi) a nephropexy is done. There were no deaths in the series, and most of the complications occurred in early cases, in which either a nephrostomy tube was used or the modern chemotherapeutic attack on tuberculosis was not developed.

Experimental Ureteric Regeneration.

A. SARRA AND E. CAINAZZO (*Urologia* (*Treviso*), October, 1959) have conducted experiments in which they removed a generous window of whole thickness of ureteric wall in four dogs, and then observed the naked eye and histological appearances after intervals of 14, 20, 35 and 50 days. At the end of 14 days periureteric fibrosis and ureteric angulation were seen, while microscopically there was "subtotal" regeneration of the epithelium, but no regeneration of the muscle layer, granulation tissue filling the gap. After 20 days the same naked eye appearances were seen, while histologically epithelial regeneration was complete, muscular ingrowth still being absent. After 35 days the peri-ureteric fibrosis had almost disappeared, and so had angulation and narrowing of the lumen; histologically muscular regeneration and closing-in was about 70% complete. After 50 days all periureteric fibrosis had completely resolved, and the lumen of the duct was well open. Histologically, epithelial regeneration was complete, and muscular reconstitution about 98% complete. Drainage by T-tube plays an important part in aiding this regeneration. It keeps the ureter straight, and allows internal drainage of urine through the lumen, and external drainage by the long limb of the T, and helps to keep urine away from contact with the regenerating gap. There is very little fear of infection provided antibiotics are used while the tube is in place, and there is no fear of phosphatic encrustation if the tube is not left too long. It was removed on the tenth day in these experiments.

Vesico-Ureteric Reflux in Children.

D. E. LEUZINGER, J. K. LATTIMER AND C. B. MCCOY (*J. Urol.* (*Baltimore*), September, 1959) have made a detailed study of 148 children with ureteric reflux. Of these, 102 had a mechanical obstruction at the vesical neck, 24 had neurogenic bladders and in 22 the condition was due to unknown causes. All those classified as having neurogenic bladders also showed other neurological defects, and most of them had meningoceles. The main conclusions of the study are as follows: (i) Untreated ureteric reflux tended to be deleterious to the kidney involved. (ii) Conservative treatment was surprisingly effective in helping this condition. (iii) The efficacy of simple urethral dilatation with sounds was surprising. (iv) When the bladder is very dilated, it affords a "cushion" which protects the ureter and renal pelvis from more severe dilatation. (v) The ordinary signs and symptoms of urinary infection, in the form of fever and pyuria, are by far the commonest symptoms in patients with reflux. (vi) All the patients who died, or failed to improve, entered the hospital with very severe dilatation of the urinary tract and elevation of the non-protein nitrogen level in the blood; failure of the azotæmia to resolve after drainage was an ominous sign.

Direct Uretero-Cysto-Neostomy.

I. J. ZIMMERMAN, W. E. FRECOURT AND C. C. THOMPSON (*J. Urol.* (*Baltimore*), February, 1960) describe a new technique for anastomosis of a short ureter with the bladder. The method is described as used in the cure of uretero-vaginal fistula, but it is suggested that it should be useful in other situations in which it solves a problem. The plan is based on the knowledge that the detrusor muscle exhibits the properties not only of contraction and relaxation, but also of elasticity; the latter property acts beyond the limits of physical relaxation. The short ureter, always a problem, has become more frequently so with the increased use of extensive pelvic surgery, in which the entire lower part of the ureter may be destroyed. The technique described brings the bladder to the ureter and, in doing so, must satisfy four important conditions. These are: (i) the

part of the bladder ready to receive the ureter must be fixed in position, thus mimicking the situation normally present; (ii) the short ureteric stump and the bladder must meet without tension; (iii) any tendency to ureteric reflux should be controlled; (iv) there should be a tendency for the bladder eventually to resume its normal anatomical relationships. The authors demonstrate their results in three successive cases of uretero-vaginal fistula, and consider that the above conditions have been satisfied. At operation the portion of bladder wall to be stretched is free from all bonds, including some of the paravesical fascia. An incision is made into this lateral aspect of the bladder reasonably high on the dome, the index finger is now passed in, and an elongated corner is made by stretching the bladder until it reaches a point on the ileo-psoas fascia beyond the tip of the fresh ureteric stump, and the tip of the corner is anchored to the ileo-psoas fascia by sutures to prevent its retraction.

Prognosis of Genito-Urinary Tuberculosis.

G. L. GALE AND W. K. KERR (*Canad. med. Ass. J.*, December 15, 1959) state that genito-urinary tuberculosis is still an important problem, but that its prognosis has been remarkably improved in the last decade. The authors review a series of 818 cases of genito-urinary tuberculosis treated under the auspices of the Toronto Hospital for Tuberculosis. The first series of 82 patients, treated without nephrectomy or chemotherapy, were followed up for 20 to 35 years, and thus represent the natural history of the disease. The second series of 347 patients, treated with nephrectomy as indicated but without chemotherapy, were followed for periods up to 15 years. The third series of 389 patients treated with chemotherapy and nephrectomy where indicated, were followed up for one and a half to 11 years. The gross mortality for the first 10 years of the disease has fallen from over 50% without chemotherapy to 4% with 12 or more months of triple drug treatment, and the incidence of deaths from renal tuberculosis has fallen from 27% to zero in the last 214 patients receiving six or more months of chemotherapy. The authors state that these phenomenal gains are largely due to drug treatment, but that in a small group of patients who formerly died of hydronephrosis due to mechanical sequelæ of healing tuberculous areas in the urinary tract, reconstructive surgical procedures such as ileo-cystoplasty have played an important part. The progress made in the management of genito-urinary tuberculosis out-ranks all other advances in urology in the past 10 years.

DERMATOLOGY.

Chondrodermatitis of the Ears.

L. P. BARKER, A. W. YOUNG AND W. SACHS (*A.M.A. Arch. Derm.*, January, 1960), in discussing chondrodermatitis of the ears, state that nodules of the helix and of the antihelix are similar only in that they appear upon the ear. The clinical course of the lesions, the underlying histological processes, and their management differ. The nodules of the helix of the ear are commoner in men over 50 years of age, whilst those of the antihelix most frequently occur in elderly women. Both types of lesion are ordinarily unilateral. The early lesions of the helix are single; those of the antihelix may be single or multiple. Nodules of the helix remain unchanged and painful throughout their course. They recur unless properly treated by surgical means. Several stages of evolution are found in the nodules of the antihelix. These are: (i) early or acute inflammatory changes with resulting ulceration; (ii) late or subacute subsidiary lesions; (iii) latent or chronic nodules detected by palpation. Acute lesions may progress to the latent variety in four or six weeks and are almost invariably associated with trauma. Only local therapy is necessary. The histology of the nodules of the helix and the antihelix is dealt with. The authors finally present a summary of the differences in appearance, location, pathogenesis, pathology and therapeutic requirements of the nodules of the helix and of the antihelix. They suggest the terms dermatochondritis helicis, because nodules of the helix arise initially in the cartilage, and chondrodermatitis antihelicis, because lesions of the antihelix arise primarily in the cutis.

Post-Operative (Pressure) Alopecia.

R. R. ABEL AND G. M. LEWIS (*A.M.A. Arch. Derm.*, January, 1960) report that sudden loss of scalp hair was observed in eight women after operation. This followed long pelvic procedures with the patient in the Trendelenburg position. A few days after operation five of the eight patients noticed œdema, exudation and crusting of the scalp on or near the vertex. One to four weeks later there appeared a patch of alopecia which resembled alopecia areata. Histopathologically the distinctive feature was an obliterative vasculitis. Regrowth of the hair occurred in from four to 12 weeks. The cause was considered to be pressure-induced ischæmia, resulting in temporary cessation of follicle activity. This theory was supported by the experimental production of alopecia in cats by a comparable mechanism.

Goeckerman Therapy.

H. N. COLE (*A.M.A. Arch. Derm.*, December, 1959) discusses Goeckerman therapy in the management of common dermatoses. After various time schedules for ultra-violet therapy had been tried, the schedule of a treatment every two weeks was finally adopted. Of the 743 patients treated, 12 showed definite sensitization to coal tar. Of the 16 different dermatoses included in this study 10 are common conditions with which the dermatologist is ordinarily confronted (psoriasis, recalcitrant pustular eruptions, nummular eczema, seborrhœic dermatitis, atopic dermatitis, neurodermatitis, psoriatic keratoderma, chronic infectious dermatitis, pustular psoriasis and dyshidrosis). The action of coal tar or ultra-violet light on the abnormal skin is still largely a matter of conjecture. The author suggests that Goeckerman therapy is an extremely useful adjunct to the treatment of many common dermatoses in addition to psoriasis, and states that it is of particular value in the management of a variety of recalcitrant dermatoses which are notoriously unresponsive to other measures, including X-ray therapy.

Trichophyton Rubrum Infection and Cushing's Syndrome.

L. M. NELSON AND K. J. MCNIECE (*A.M.A. Arch. Derm.*, December, 1959) report the case of a patient with Cushing's syndrome and a *Trichophyton rubrum* infection. Despite treatment, the infection continued to spread until the patient was subjected to subtotal adrenalectomy, after which his general condition showed great improvement and the fungous infection almost cleared. Two and a half years later the symptoms of hyperadrenalism returned and there was a severe exacerbation of the fungous infection. The patient then had his remaining adrenal tissue removed and was placed on total substitution therapy. This was followed by the disappearance of his general symptoms, and the *Trichophyton* infection promptly subsided to minimal involvement of nails and adjacent skin.

Systemic Treatment of Atopic Eczema with Steroids.

I. B. SNEDDON (*Brit. J. Derm.*, January, 1960) gives an account of continuous systemic administration of steroids to 26 adults suffering from severe atopic eczema. Treatment has been maintained for periods of four months to four and a half years. Control of symptoms has been good in 19 cases, fair in five, poor in two. No difference in efficacy has been noted between dexamethasone and prednisolone. Final maintenance doses have been in the region of 10 to 15 mg. of prednisolone daily. No serious side effects have been encountered, but the majority of patients gained weight and developed moon face. It is stressed that some harmful effects may yet arise and treatment should not be embarked upon lightly or in the absence of adequate facilities for close supervision.

Melanotic Freckle.

M. J. COSTELLO, S. B. FISHER AND C. P. DE FEO (*A.M.A. Arch. Derm.*, December, 1959) state that in 70% of cases of melanotic freckle the lesion involves the face. Melanotic freckle is an easily recognizable pigmented pre-cancerous macule which is frequently seen on the face of patients over 40 years of age. It usually exhibits a histo-pathological picture resembling a pre-malignant junction nævus, which can slowly progress to a tumour stage showing histological features of malignant melanoma. Individual treatment consists of surgical excision or electrodesiccation and curettage, and these will provide good therapeutic, cosmetic and functional results. Ten illustrative cases are described. The authors state that long-term observation of patients with this clinically distinctive lesion is needed to corroborate the impression that the prognosis in a patient with a melanotic freckle is more favourable than that in a patient with classical malignant melanoma.

Clinico-Pathological Conferences.

A CONFERENCE AT SYDNEY HOSPITAL.

A CLINICO-PATHOLOGICAL CONFERENCE was held at Sydney Hospital on July 21, 1959. DR. H. M. WHYTE was in the chair, and the principal speaker was DR. A. E. MCGUINNESS, Honorary Physician to the hospital.

Clinical History.

The patient, a farm-hand, aged 47 years, complained of occasional attacks of diarrhœa for five months, and of recurrent attacks of epigastric pain (lasting several hours and initially relieved by A.P.C.) for two months. There was occasional vomiting, and he had passed very dark stools. He had previously been well, apart from occasional palpitations at night time, an appendicectomy in 1925 and a painful left hip joint in 1943. His mother had died at the age of 74 years from "blood pressure" and aortic aneurysm; otherwise his family history was not relevant. He smoked about 5 oz. of tobacco per week and drank little alcohol. He was admitted to a country hospital after an attack of abdominal pain and vomiting. X-ray examination with a barium enema and a chest X-ray film revealed no abnormality; a barium-meal X-ray examination revealed only marked œsophageal reflux. The hæmoglobin value was 7 grammes per 100 ml. and the leucocytes numbered 15,000 per cubic millimetre; the serum bilirubin content was 4·5 mg. per 100 ml.; 5·6% of the erythrocytes were reticulocytes; osmotic fragility ranged from 0·48% to 0·34%. After a recurrence of severe abdominal pain and vomiting, intestinal obstruction was diagnosed and laparotomy was performed. A large retroperitoneal hæmatoma was found arising in the region of the right kidney and enveloping the ascending colon. He was then transferred to Sydney Hospital.

On his admission, he complained of persistent severe abdominal pain and was vomiting. He was a lean man with a dark skin slightly jaundiced. His blood pressure was 200/130 mm. of mercury, his pulse rate was 120 per minute and he had intermittent elevations of temperature to 100° F. The abdomen was tense, but not tender. The right paramedian laparotomy wound was healing well. There was a hard mass of regular contour in the right iliac fossa. Neither the liver nor the spleen was palpable. The bowel sounds were normal; the fæces were pale, somewhat yellow, but well formed. The right inguinal lymph nodes were enlarged, and small nodes were palpable in each axilla. The venous pressure was raised one inch. The apex beat was in the fifth left intercostal space at the anterior axillary line. A systolic murmur, heard in all areas, was thought to arise at the mitral valve. Respiratory movement was poor at the base of the left lung, with an impaired percussion note and marked pleural friction. Examination of the optic fundi revealed Grade III hypertensive retinopathy. The urine contained a trace of albumin, but no bile. An X-ray examination of the chest revealed patchy consolidation and fluid at the base of the left lung. A number of tests were carried out, with the following results: hæmatocrit, 33·5%; normochromic erythrocytes; 20,800 leucocytes per cubic millimetre (88% neutrophils, 1% eosinophils, no basophils, 7% lymphocytes, 4% monocytes, abnormal mononuclear cells, an occasional myelocyte); 171,000 platelets per cubic millimetre; coagulation time (Lee White), 5 minutes; prothrombin index 73%. The direct Coombs test produced a negative result. The blood urea nitrogen content was 40 mg. per 100 ml. The serum protein content was 6·0 grammes per 100 ml. (albumin 2·2, globulin 3·8). The thymol and zinc turbidity were respectively 2 and 7 units. The serum alkaline phosphatase content was 39 King-Armstrong units. The van den Bergh test produced a faint direct positive reaction. The serum bilirubin content was 1·1 mg. per 100 ml. Microscopic examination of the urine revealed 30 to 50 erythrocytes per high-power field and an occasional leucocyte. A bromsulphalein test showed that 17% was retained after 45 minutes (5 mg. per kilogram). There was no occult blood in the fæces. Inguinal lymph-node biopsy revealed dilated sinuses containing fresh erythrocytes, and sinus catarrh. The proportion of plasma cells was increased.

After laparotomy, abdominal pain and vomiting continued; the patient became obviously jaundiced, and bile was present in the urine. The serum bilirubin content was 2·5 mg. per 100 ml., and the van den Bergh reaction was positive. The hæmoglobin value fell to 5·5 grammes per 100 ml., the proportion of reticulocytes being 24%. The erythrocytes had a full content of hæmoglobin; occasional spherocytes, marked anisocytosis and moderate microcytosis were present. The number of platelets fell to 33,000 per cubic millimetre, a widespread purpuric rash appeared and the result of the Hess test was positive. L.E. cells were not found. Examination of the bone marrow revealed normoblastic hyperplasia of erythropoiesis. Five milligrammes of "Regitine" given intravenously reduced the blood pressure from 150/120 to 120/90 mm. of mercury for six minutes, and when the dose was repeated, from 185/135 to 140/110 mm. of mercury for 20 minutes. The liver was now palpable three fingers' breadth below the costal margin, and the patient had ascites, congestive failure and attacks of acute pulmonary œdema. As well as symptomatic treatment, he was given digitalis, "Chlotride", prednisone and repeated blood transfusions. His condition improved sufficiently to allow his return to the country, where he died shortly afterwards.

Clinical Discussion.

DR. A. E. MCGUINNESS: This is a case of interest, in so far as it concerns a male, aged 47 years, who suffered a fulminating illness and had two laparotomies which I believe were not themselves diagnostic of his pathology. The illness was of brief duration. The initial presentation was of gastro-intestinal symptomatology with recurrent melæna. Later the negative occult blood studies would suggest that he suffered episodic bleeding from the gut.

At his first hospital admission there is evidence of hæmolytic anæmia and a retroperitoneal hæmatoma. At Sydney Hospital the diagnosis of hæmolytic anæmia was established, and finally he suffered a further retroperitoneal hæmorrhage in the right iliac fossa, and showed evidence of severe hypertensive disease with renal involvement and a complicated hepatic and pulmonary pathology.

After an illness of a few months he perished, probably from cardiac failure and complicating anæmia. I believe the significant features of this illness to be a widespread vascular pathology in the presence of a hæmolytic anæmia. Hæmolytic anæmia is substantiated by blood counts at this hospital showing a normocytic normochromic anæmia, and finally a microcytic normochromic pattern with evidence of spherocytosis. Throughout the illness the hæmoglobin was of a low level, and the reticulocyte count was elevated. Initially a reticulocyte count of 5·6% was found in the presence of a serum bilirubin level of 4·5 mg. per 100 ml. Dameshek has pointed out succinctly that although other causes may be responsible for a reticulocyte count of 5%, hæmolytic anæmia must always be considered in its presence. I believe these findings support a diagnosis of hæmolytic anæmia at this time.

It may be argued that, in the presence of a hæmatoma within a serous cavity, the serum bilirubin may be accounted for by this circumstance; but I believe this level to be too high for this explanation to be accepted.

The white-cell count is compatible with the presence of a hæmolytic anæmia; but the sustained level of the leucocytosis and the final leukæmoid reaction suggest that some other underlying pathology was present to account for these findings. A leukæmoid picture such as this suggests a severe infection, such as septicæmia and possibly subacute bacterial endocarditis etc. The low intermittent fever and the absence of splenomegaly and signs of embolic phenomena would tend to exclude both these diseases. A leukæmoid reaction is not uncommonly encountered in hepatic disease, especially in hepatic carcinoma. I will endeavour to exclude this diagnosis later, and I do not believe that it was a feature of this illness.

Again, Talbot has noted this degree of leucocytosis in thrombotic thrombocytopenic purpura, as also has McGhee Harvey in his study of disseminated lupus erythematosus. A mesenchymal disorder may be the solution to this aspect of the illness. The platelet count is significant. Initially he presented with a normal platelet count, which fell precipitously during the course of the illness. A diagnosis of splenism or hypersplenism cannot be substantiated in view of the leucocytosis and absence of splenomegaly.

Neoplastic involvement of the marrow may be suggestive; but marrow biopsy failed to show evidence of neoplastic infiltration, and this finding tends to be confirmed by the lymph-node biopsy, which failed to show evidence of neoplasia. Therefore, although the absence of neoplasia on one biopsy is slim evidence, I believe that marrow infiltration is not the cause of this thrombocytopenia. I am suggesting at this stage that the thrombocytopenia is due to his hæmorrhagic disorder, which was an essential feature of this illness. I believe the liver to be the key

to the diagnosis of this patient's illness, and will next consider the hepatic investigations. The bromsulphalein test showed 17% retention at a time when the jugular venous pressure was elevated, and this complication raises doubt as to the significance of this degree of retention in this case. The test depends upon an intact circulatory state for diagnosis of hepatic parenchymal cell damage.

Again, the protein findings are perhaps not as significant as they appear, when it is realized that this man was suffering a severe constitutional disturbance in which the hyperglobulinæmia may merely reflect a generalized metabolic disturbance. Similarly, a fall in the serum albumin may occur in such a circumstance. These facts are significant in the assessment of these studies, but I believe that the following conclusions can be established. (i) There is no evidence for a diagnosis of extrahepatic obstruction; the serum bilirubin level of 1·1 mg. is incompatible with this diagnosis in the presence of a serum alkaline phosphatase level of 37 units. (ii) Although the bromsulphalein level is raised, as is the serum alkaline phosphatase level, in the presence of a normal serum bilirubin, and is therefore suggestive of hepatic infiltration by neoplasm, sarcoid, tuberculosis or leukæmia, this assumption can be accepted solely if the concept is accepted that the protein tests are themselves representative of a severe generalized disturbance rather than of hepatic involvement alone. If one argues that they represent a true metabolic disturbance of the liver secondary to hepatic-cell injury, then they are compatible with a pathology causing hepatic-cell necrosis. I believe that the size of the liver itself is significant to this discussion. If infiltration is the cause of this hepatic disturbance, one would expect an enlarged liver. The liver was said to be impalpable, and I believe that this finding suggests that infiltration was not the explanation for these findings.

With a presumptive diagnosis of mesenchymal-cell damage, do these tests suggest a cirrhotic process or a hepatitis? A cirrhotic liver is usually enlarged or sometimes small. A viral or toxic hepatitis is unlikely in the presence of leucocytosis and bleeding phenomena, and the history suggests that a severe hepatic disturbance is occurring and one must determine its ætiology. The history of the illness, with the two hæmatomas, the purpura and the bleeding tendency, suggests that a similar disturbance is occurring in the liver, which I believe to be due to arterial thrombosis, hæmorrhagic phenomena and ischæmic mesenchymal-cell necrosis. Further evidence for this conclusion was obtained indirectly from the lymph-node biopsy. This biopsy disclosed the presence of histiocytes, plasma cells and erythrophagocytosis. Marchant, in the *Annals of Internal Medicine* in 1954, described lymph-node erythrophagocytosis as a common finding in malignant hypertension and severe hypertension of renal origin. It occurs also in nodes draining vascular tumours and lymphomata. The common denominator to all these conditions is hæmorrhage, and I believe that this node finding in its anatomical relationship to the right iliac fossa suggests that the mass in the right iliac fossa was a hæmatoma. This opinion is based on the absence of evidence of leukæmia, lymphoma and neoplastic infiltration in the node biopsy. Acute medullary histiocytic reticulocytosis as a cuase for this finding does not require consideration.

The renal tests disclose significant hæmaturia and azotæmia and are compatible with severe renal involvement, probably of the smaller vessels. The X-rays are significant. The first chest X-ray was reported as "clear" and so renders a diagnosis of tuberculosis unlikely. Subsequent X-rays as presented today are diagnostic of a Löffler's pneumonitis, and hence establish my premise more firmly. The X-rays of the gastro-intestinal tract are just as significant. There is no evidence of intrinsic carcinoma or ileitis, this fact tending to be confirmed by the negative occult blood studies. I find, therefore, little to suggest intra-abdominal neoplasm. More significantly, the absence of extensive pressure effects on the gastro-intestinal tract in these studies adds further confirmation of the absence of extensive masses, as might be found in abdominal neoplasm. Admittedly, carcinoma of the colon or stomach can present with widespread metastases and a minimally-sized primary lesion, but the X-ray studies do not suggest the presence of a mass in the abdomen.

Thus, I believe that these investigations point to a hæmorrhagic disturbance in the presence of a hæmolytic anæmia. Turning to the clinical solution, on arrival at Sydney Hospital, a tense, painless abdomen on palpation is described in the presence of recurring attacks of pain. Previously he experienced diarrhœa and recurrent malæna and a laparotomy, the indication being intestinal obstruction. I believe this history to represent recurrent small-vessel pathology, and that at Sydney Hospital he was suffering from a bloody ascites. Later this became apparent. The unusual radiation of the murmur from the apex to the base is not explained convincingly by the circulatory hæmodynamics attending upon anæmia and fever. In the presence of a vascular pathology, Libman-Sachs endocarditis, a complication of disseminated lupus erythematosus, must be considered. In McGhee Harvey's series, Libman-Sachs was noted in 40% to 50% of cases, but these investigations do not produce murmurs commonly. Admittedly the murmur may represent the exuberant proliferating vegetation as noted by him to occur in 10% of cases.

My diagnosis in this case, therefore, is one of a hæmorrhagic disorder associated with glomerulo-nephritis, a widespread vascular visceral involvement as suggested by episodic bleeding from the bowel, and two major hæmatomata, one retroperitoneal and the other in the right iliac fossa. These findings point to the mesenchymal disorders, and the sustained hypertension, severe degree of leucocytosis and the continuous fever suggest that the diagnosis is polyarteritis nodosa. This disease rarely presents with hæmolytic anæmia, but kindred diseases, such as thrombotic thrombocytopenic purpura and disseminated lupus erythematosus, may present with this complication. In Harvey's series this complication was noted in 5%. The lungs will probably show evidence of a vascular pneumonitis (Löffler's pneumonia), and the liver arterial thrombosis with parenchymal-cell necrosis and hæmorrhage. The kidneys will show evidence of this disease, but the expression of it is speculative, pathologically. The response to steroids is in keping with this diagnosis as, in the presence of renal involvement, a remission is transitory and not followed commonly by a sustained period of recovery. I believe that neoplasm will not be confirmed at autopsy. Two factors are against this diagnosis. The early presentation of a hæmolytic anæmia is unusual in carcinoma. Furthermore, a hæmorrhagic diathesis is uncommon with neoplasm and when present points to chorionic epithelioma, prostate, lung or pancreas as the primary neoplasm. This presentation would be unusual.

Hepatoma warrants consideration before dismissal, as it is known that it is commonly associated with cirrhosis of the liver. King has shown that pancreatitis is not uncommon as a complication of cirrhosis. Again, Patch, in studies of autopsied subjects with cirrhosis, noted a 10% incidence of glomerulonephritis. I will conclude with a diagnosis of periarteritis nodosa with a rare complication of this disease, acquired hæmolytic anæmia.

DR. H. M. WHYTE: Well, the puzzle is solved: or is it? It allows of a great deal of scope for much argument and discussion—hæmolytic anæmia, glomerulonephritis, bleeding, high blood pressure, leucocytosis, involvement of liver, lungs and heart, and fever—that is what Dr. McGuinness has taken as a basis for his diagnosis of polyarteritis nodosa, with the unusual accompaniment of hæmolytic anæmia. Who would like to comment on any of these aspects?

DR. R. J. WALSH: I would like to make some comments on a few points Dr. McGuinness has raised.

The first concerns the reticulocytosis. If 1% of the erythrocytes in the blood sample with 5,000,000 cells per cubic millimetre are reticulocytes, then the same numbers of reticulocytes are present in a sample with 5% reticulocytes in a total of 1,000,000 cells per cubic millimetre. Both reticulocyte percentages indicate the same degree of marrow activity. I would make a plea, Sir, that one should not consider reticulocytosis as a percentage of the red cells, but that one should think of the absolute number. One then has an idea of what 24% or 5·6% reticulocytes means, and an idea of whether the bone marrow is working at its normal rate or trying to compensate for a hæmolytic process. With regard to the leucocytosis, Dr. McGuinness mentioned that this was often associated with hæmolytic anæmia and was known as a leukæmoid reaction. I think that a leucocytosis always occurs when there is excessive bone marrow activity. In other words, the myeloid tissue multiplies when the erythroid tissue is multiplying rapidly. This leucocytosis is therefore a non-specific response, not to hæmolysis or hæmorrhage, but to marrow activity following hæmolysis or hæmorrhage. The presence of an occasional myelocyte in such a patient is, I feel, of no significance, and merely indicates over-activity of the myeloid tissues.

The question of thrombocytopenia in patients with liver disease is an interesting one, as it is not uncommon.

Some years ago, Swedish workers showed that implantation of liver under the skin or injections of alcoholic extracts of liver produced a thrombocytosis in rabbits. They suggested that the liver may control the level of platelets in the blood, with the production of thrombocytopenia in liver failure. Is this the explanation in the case being discussed? Is it perhaps part of Dr. McGuinness's multi-system disease?

The albumin-globulin ratio is also of interest. Is it a specific increase of the gamma globulin? Recent work has shown that gamma globulin is the only plasma protein which is not synthesized by the liver. Is the increase in globulin related to infiltrative disease or other abnormality of the liver?

DR. WHYTE: Before anybody answers all your questions, I wonder if you would make it clear to me whether you are suggesting the marrow is over-active or is not. On the score of the white-cell count you suggested it was over-active; but, on the score of the reticulocytes, did you suggest it was normally active?

DR. WALSH: No. I think 24% reticulocytosis with 5·5 grammes of hæmoglobin suggests over-activity of the erythroid disease. My plea was for an expression of the reticulocytes in absolute numbers. In this case I think the marrow was over-active in the later stages, although it may not have been so in the beginning.

DR. K. B. NOAD: In thinking and talking about this problem, I came to much the same conclusion as the speaker, that here we had a number of systems involved, and thought that this was, as he said, evidence of another mesenchymal disorder, probably polyarteritis nodosa. But I notice that Dr. McGuinness did not say anything about the "Regitine" test, at least not while I was here. After all, I know that it is suspect and not thought to be perhaps of very great value; but, however, if it is of any use, it gave a positive response, and that gave one cause to wonder whether perhaps malignant disease was in fact present here. Naturally we would associate that malignant disease with the adrenal, and the protocol did suggest a progressive story with enlargement of the liver and a rise in the serum bilirubin and ascites and so forth. However, on second thoughts, perhaps these phenomena could be explained by the progressive heart failure which appears to have developed in this man, because it says the liver was palpable, there was ascites and he developed congestive failure with attacks of acute pulmonary œdema. So, perhaps these progressive changes noted in the physical examination in the abdomen could be explained on the basis of progressive heart failure and not on the basis of metastases from a neoplasm. And so those are the two conditions that entered into my thoughts about this case, and of the two I think I favour polyarteritis nodosa, as Dr. McGuinness did.

DR. MCGUINNESS: I believe the "Regitine" test to be a red herring. It should be appreciated that the "Regitine" test may be falsely positive in the presence of renal hypertension and barbiturate administration, and I believe the finding in this case is misleading.

DR. R. J. ELVY: I would like to support Dr. Walsh in his plea for describing reticulocytes in terms of absolute numbers, but I would like to extend that a bit further in regard to the problem of establishing hæmolysis with reticulocyte counts. Certainly one reticulocyte count is of very little value, and the only thing to do is to follow patients regularly with multiple reticulocyte counts, and then if reticulocytes do represent constantly more than 2% of the circulating red cells, irrespective of their total numbers, in the presence of a static or falling hæmoglobin or hæmatocrit, then this almost certainly represents increased red-cell destruction, provided that blood loss is excluded. Well, in this patient that was done and he had a very high percentage of reticulocytes throughout this episode of several weeks, and his hæmatocrit never rose above the level of about 30%, which is approximately 10 grammes of hæmoglobin. As regards the problem of hæmolysis in this patient, I think it was established on that basis and on the basis of the film abnormality, which showed persistent evidence of the pattern seen in hæmolytic anæmia. He also had spherocytosis and neutrophilia, both of which are a not uncommon accompaniment of severe hæmolytic anæmia; thrombocytopenia is again a not uncommon accompaniment of acquired hæmolytic anæmia, but I cannot say any more on this point without disclosing the diagnosis.

DR. E. HIRST: Mr. Chairman, do we have any information on Dr. Walsh's question about the gamma globulins in this case?

REGISTRAR: No, we have no electrophoretogram.

DR. J. RAFTOS: I know the diagnosis, as I looked after this man, so that I will not discuss the case; but I will take issue with Dr. McGuinness's interpretation of the chest X-rays as showing Löffler's syndrome. I think that this is a term that is very loosely used. If Dr. McGuinness means merely a chest shadow which alters fairly rapidly, well and good; but to me Löffler's syndrome means something very much more specific. My interpretation of these shadows during life was that they were in fact pulmonary infarction due to either a local thrombotic process or an embolus.

DR. MCGUINNESS: The diagnosis of Löffler's pneumonitis may have been confirmed by the finding of eosinophilia in the sputum. It is not an uncommon manifestation of polyarteritis nodosa, and the X-rays presented today show quite clearly the transitory pneumonitis so characteristic of this disease. I use the term Löffler's syndrome, as not all presentations of Löffler's pneumonitis are due to polyarteritis ætiologically; others are eosinophilic granuloma of the lung and tropical eosinophilia, for example, and present in this way radiographically.

DR. B. P. BILLINGTON: I am confused. Everyone is talking about the bone-marrow reaction which is going on with the hæmolytic anæmia. All of a sudden towards the end of the illness the platelets just fizzle out completely. I cannot quite understand this on any explanation that has been put forward, except possibly on the grounds either that there is an immune process involved, or that there is bone-marrow replacement by abnormal tissue.

DR. WHYTE: Dr. Walsh has suggested that if he were an experimental animal and had had some liver put under his skin, that could have accounted for it; but he has not, of course.

DR. B. M. HURT: Like Dr. Billington, I regard the protocol with some suspicion when the bone-marrow results are given only on the red-cell side. There is no mention of white cells or platelets. I wondered originally whether this man had a disturbance of his proteins as in a pre-malignant condition and his 3·8 to 2·2 ratio of globulins to albumin might represent a macroglobulin problem. I understand that these people can get hæmorrhages and purpura, and it might be reflected in the bone marrow with abnormal leucocyte production. It could also explain his hæmolysis and his severe anæmia. I think the clinical side has been a little confused about his systolic murmur thought to arise at the mitral valve. Here is a very sick man with anæmia, hypertension and cardiac failure, and this systolic murmur might not reflect an organic problem at the valve. Then the fundi showed Grade III hypertensive retinopathy. I wondered whether this indeed was just hæmorrhages in his retina. It has been suggested that globulin disturbances can precede malignant disease by some years, and I wonder, indeed, whether this man was not building up to some malignant condition such as in his adrenal glands or lung, although there seems to be no problem in his lung which has progressed. I rather tend to agree with Dr. Raftos that there is one problem in his right upper lobe and also pulmonary œdema, which I see from the protocol was overcome with digitalis and "Chlotride". I would have regarded that bottom left-hand film as just showing pulmonary œdema. However, I can not elucidate the primary diagnosis. I would like to know whether the bone marrow shows more evidence.

REGISTRAR: That was the only abnormality in the bone marrow. There were good fragments obtained which were of increased cellularity. Erythropoiesis showed normoblastic hyperplasia with a number of macronormoblasts and occasional binuclear cells. Leucopoiesis was normal. There was an occasional abnormal metamyelocyte and hypersegmented neutrophil. Lymphocytes were present, but not increased; megakaryocytes fairly plentiful; reticulum cells present; plasma cells not increased; no abnormal cells; iron stores were plentiful and mitoses were present.

DR. MCGUINNESS: As regards macroglobulins, they are occasionally found in malignancy. The disease as described by Waldenström presents a benign or malignant form. The benign form is characterized by purpura and lymphocytic infiltration of the marrow. The malignant presentation is that of adenopathy, hepatomegaly, splenomegaly, anæmia and again lymphocytic infiltration of the marrow. This patient's findings do not conform to these requirements for a diagnosis of this state. Hyperglobulinæmia occurs commonly in many infectious hepatic disorders, and especially in neoplasia of the reticulo-endothelial cell system. Recently I had under my supervision a patient who I believe has a dysgammaglobulinæmia. She

suffered eleven attacks of pneumonia in four years, and her gamma globulin was 13 grammes per 100 ml. This was the only abnormality that was established in the studies. This history is relevant to the point that Dr. Hurt raised—what happens to this group of subjects who apparently are producing abnormal gamma globulin? In any case, in answer to Dr. Hurt, a diagnosis of macroglobulinæmia cannot be substantiated in the absence of electrophoretographic studies.

Autopsy Findings.

DR. A. A. PALMER: Post-mortem examination was made in the country, and we are most grateful to the doctor for sending the protocol and tissue for histological study. There were pleural effusions and the lungs were œdematous. The heart was moderately enlarged. In the liver there was a round, firm, pale yellow nodule 5·5 cm. in diameter with central hæmorrhage and necrosis; elsewhere the cut surface of the liver was congested and mottled. The spleen was slightly enlarged. Round or ovoid nodules with fibrous capsules and centres of dark-red tissue resembling blood clot were found in the mediastinum (1·0 cm.), near the pylorus (0·6 cm.) and near the pancreas (1·5 cm.). The right kidney had a firm hemispherical nodule 4 cm. in diameter projecting from its upper pole, and other smaller nodules were present in the renal substance, all with hæmorrhagic centres. The kidney showed some congestion and scarring. Below and medial to the right kidney there was an old encysted retroperitoneal hæmorrhage.

Sections of the liver show arteritis; many of the lesions are healed, often with fibrous occlusion of the vessel, but there are several still-active lesions. The nodule is an infarct, and there is much other evidence of circulatory disturbance. Sections of the nodules from the region of the pylorus, mediastinum, pancreas and kidney show that they are aneurysms filled with old partly organized clot. These were presumably sites of former arteritis, but direct evidence for this is now mostly absent. In the kidney there are also fairly severe changes of hypertensive nephrosclerosis. There is also healed arteritis in the adrenal and prostate. The spleen shows abundant iron-containing pigment consistent with hæmolysis.

Pathological Discussion.

DR. WHYTE: Dr. McGuinness is to be congratulated on making this diagnosis as, too, are the other speakers who arrived at the same conclusion. It is obviously a very terrible and terrifying disease that can demolish a comparatively young man like this in such a short time. Perhaps, very briefly, Dr. Raftos might like to tell us how this diagnosis can be established during life, and whether there is any evidence that in our present state of knowledge any treatment is of any avail.

DR. RAFTOS: This case was most interesting. I looked after this man in Dr. Fisher's temporary absence last year, and it happened that two weeks before he was admitted we had a Greek lady admitted with a most peculiar abdominal syndrome. She had a laparotomy, and she had polyarteritis nodosa, of which she died very rapidly. At this stage, in discussion with Dr. Hirst, he suggested it might be an idea to do a biopsy of the rectal mucosa, making sure that we got some submucosa as well. This was done in her case, and polyarteritis was confirmed. So it seems that this might be a valuable method of investigation in these cases. In this patient's case we treated him as having polyarteritis nodosa and we gave him the corticoids with, I think, an improvement lasting about two weks, after which his condition deteriorated quite rapidly. The hæmolytic anæmia was an unusual feature; but at the present time we have a boy in hospital who presents very much the same picture, so that it might not be quite as rare as we would anticipate. This is a boy with recurrent thrombophlebitis, who, on his last admission, had an acquired type of hæmolytic anæmia, and who is now developing some degree of hypertension. He is getting polyarthralgia as well, and I think that he will probably turn out to have a disease such as this.

Well, the diagnosis can only be made firstly by clinical analysis, as Dr. McGuinness did, and then confirmed by biopsy. I think in any disease in which there is evidence of either thrombosis or hæmorrhage, the diagnosis of polyarteritis has to be considered very strongly. When, in addition, there is evidence of hypertension which develops quite rapidly, then the diagnosis can be made on quite firm clinical grounds.

Diagnosis.

Polyarteritis nodosa with small aneurysms in the mediastinum, retroperitoneal region and kidney, retroperitoneal hæmorrhage, hypertension and nephrosclerosis, hæmolytic anæmia and thrombocytopenia.

British Medical Association.

TASMANIAN BRANCH: ANNUAL MEETING.

THE annual meeting of the Tasmanian Branch of the British Medical Association was held at the Royal Hobart Hospital on March 12, 1960, Dr. L. H. WILSON, the President, in the chair.

ANNUAL REPORT OF THE COUNCIL.

The annual report of the Council, having been circulated to members, was taken as read and received. The report was adopted on the motion of Dr. L. H. Wilson, seconded by Dr. A. McL. Millar. The report is as follows.

The Council has pleasure in presenting the annual report for the year ended December 31, 1959.

Membership.

The membership is 262 members against 260 members for 1958, a gain of two.

Obituary.

It is with regret that we record the death of the following member: Dr. E. Fabian.

Meetings.

The annual general meeting of the Branch was held on March 7, 1959, at the Royal Society rooms, at which there were 40 members present.

The following office bearers were elected:

President: Dr. L. H. Wilson.

Vice-President: Dr. W. W. Wilson.

Medical Secretary: Dr. K. Melville Kelly.

Honorary Treasurer: Dr. K. J. Friend.

President Elect: Dr. R. A. Lewis.

Committee members elected were: Dr. D. B. Nathan, Dr. A. J. M. Dobson, Dr. K. S. Millingen, Dr. M. W. Fletcher.

One special meeting was held during the year, but all other business was carried out at the monthly meetings of the Southern and Northern Subdivisons.

Branch Council.

Thirteen meetings of the Branch Council were held during the year, 11 in Hobart and two in Launceston. Additional members of the Council were:

Chairman of Southern Subdivision: Dr. A. Corney.

Chairman of Northern Subdivision: Dr. R. A. Godfrey-Smith.

Secretary of Southern Subdivision: Dr. J. Correy.

Secretary of Northern Subdivision: Dr. W. Hill.

Federal Council Representatives: Dr. F. R. Fay, Dr. L. N. Gollan.

Immediate Past President: Dr. A. McL. Millar.

The following attendances were recorded:

Dr. L. H. Wilson	13
Dr. W. W. Wilson	13
Dr. K. M. Kelly	12
Dr. K. J. Friend[1]	8
Dr. F. R. Fay	13
Dr. L. N. Gollan	11
Dr. J. Correy	11
Dr. A. Corney[1]	9
Dr. R. A. Godfrey-Smith[1]	9
Dr. W. H. Hill	13
Dr. R. Hudson[2]	2
Dr. A. McL. Millar	12

[1] Elected March, 1959.

[2] Retired March, 1959.

Dr. R. A. Lewis	12
Dr. M. W. Fletcher	12
Dr. H. M. Fisher[2]	3
Dr. H. Gatenby[2]	0
Dr. D. B. Nathan	10
Dr. A. J. M. Dobson	10
Dr. K. S. Millingen[1]	8

Representatives and Subcommittees.

Federal Council.

The Branch was represented on the Federal Council by Dr. L. N. Gollan and Dr. F. R. Fay, both of whom attended interstate meetings on behalf of the Branch. We wish to record the thanks of the Branch for the way in which these members carried out their duties, and for the clear way in which they were always able to relate business and events for the benefit of Council and Subdivisional meetings.

Summary of the Most Important Business of the Year.

Annual Representative Meeting.

Dr. T. Giblin and Dr. H. Gatenby were appointed to represent the Branch at the Annual Representative Meeting in Britain in July, 1959.

Minister for Health.

Subjects discussed with the Minister for Health included doctors' fees for ante-natal care, amendments to procedure in adoption of infants, and views were exchanged on amendments to the *Medical Act* and the disciplinary powers of the Medical Council.

Medical Council.

Discussions were held on the disciplinary powers of the Medical Council. Certain complaints were referred to the Medical Council for its jurisdiction.

Health Department.

Discussions were held with the Department officers on the form of registration of births and an appended statistical record. As a result of the support given by the profession, it is expected that some results of the investigation will be available in 1960.

Several other discussions were held. The subjects were of a minor nature, but it is pleasing to record a spirit of cooperation and understanding of mutual problems between the Department and the Association.

Salaried Medical Practitioners Society.

This Society was formed towards the end of 1959, in order that salaried practitioners should be represented on the new State Tribunal formed to fix salaries. Branch Council was asked for advice on certain aspects of the society, and it is possible that a move may be made to make the society a special group within the Association.

Medical Benefits.

Several suggestions for amendments to the schedules of benefits were made and forwarded to the Commonwealth for consideration. Certain medical benefits organizations were approached concerning suggested amendments to their rules.

Medical School in Tasmania.

Representatives gave evidence before the Select Committee of the House of Assembly on the formation of a medical school.

Repatriation Department.

Discussions were held concerning some misunderstandings on after-hours facilities at the Repatriation Hospital, and the procedures were clarified. Fees for filling in various forms were discussed and opinions forwarded to Federal Council, where satisfactory action was taken.

Hobart City Council.

The Hobart City Council requested opinions and information on the question of fluoridation of the city water supply, and these were given by Council.

The attention of the City Council was drawn to the lack of public conveniences in Hobart, and the unsatisfactory state of the existing facilities provided.

[1] Elected March, 1959.

[2] Retired March, 1959.

British Medical Association House.

A building at 129, Davey Street, was purchased for £9000, to be used as an headquarters for the Branch. An appeal to members was launched for funds to convert this building, at present let as flats, and it is hoped that a start will be possible within two years.

Dentists.

Discussions were held with representatives of the Australian Dental Association on the question of dentists using the title "doctor", and eventually a compromise was agreed upon.

Chemists.

A discussion was held between representatives of the Branch Council, Pharmacy Board and Pharmaceutical Guild. This was the first time an opportunity had arisen for the exchange of views. Subjects included *Dangerous Drugs Act, Poisons Act*, prescribing and general relations. It was felt that further meetings should be arranged at intervals, and that such meetings would result in closer cooperation between the two professions.

Medical Benevolent Fund.

The Medical Benevolent Fund was operated during the year for the first time since 1940. This Fund was started in 1932 to assist doctors or their families in financial need. The balance in hand was not great, but has been used for the purpose intended.

Representatives.

The Branch was represented on outside bodies as follows:

Australasian Medical Publishing Company Ltd.: Dr. W. E. L. H. Crowther.

Road Safety Council of Tasmania: Dr. F. Phillips.

Federal War Relief Fund 1914-1918: Dr. B. Hiller, Dr. F. W. Fay and Sir Ralph Whishaw.

Federal War Relief Fund 1939-1945: Dr. R. A. Godfrey-Smith, Dr. T. Giblin and Dr. Franklin R. Fay.

Tasmanian Physiotherapists' Registration Board: Dr. J. Law.

Tasmanian Health Education Council: Dr. H. B. C. Houston.

Committees.

Ethics: Dr. R. A. Lewis, Dr. J. B. G. Muir, Dr. Franklin R. Fay, Dr. L. N. Gollan, Dr. M. W. Fletcher and Dr. W. W. Wilson.

Newsletter: Dr. J. F. Correy, Dr. K. J. Friend and Dr. K. M. Kelly.

Publicity: Dr. L. H. Wilson, Dr. R. A. Godfrey-Smith, Dr. Franklin R. Fay and Dr. A. McL. Millar.

Workers' Compensation: Dr. Franklin R. Fay, Dr. A. O. Green and Dr. A. McL. Millar.

Medical Fees: Dr. L. N. Gollan, Dr. W. H. Hill, Dr. A. C. Corney, Dr. J. B. G. Muir, Dr. F. R. Fay, Dr. A. J. M. Dobson and Dr. L. H. Wilson.

Building: Dr. F. W. Fay (chairman), Dr. D. J. Walters, Dr. Franklin R. Fay, Dr. A. O. Green, Dr. A. McL. Millar, Dr. K. Melville Kelly and Dr. H. M. Fisher.

K. MELVILLE KELLY,
Honorary Medical Secretary.

FINANCIAL STATEMENT.

The financial statement was adopted on the motion of Dr. K. J. Friend, seconded by Dr. F. W. Fay.

Dr. Friend said that donations towards the Building Fund were not up to expectations, but that he hoped that the brochure prepared and circulated would induce members who had not yet made a donation to respond.

Dr. F. W. Fay said that the finances of the Branch were in a very favourable condition.

ELECTION OF OFFICERS.

It was anounced that the officers of the Branch for 1960 were as follows:

President: Dr. R. A. Lewis.

President Elect: Dr. H. J. C. Engisch.

Vice-President: Dr. W. W. Wilson.

Honorary Treasurer: Dr. K. J. Friend.

BRITISH MEDICAL ASSOCIATION (TASMANIAN BRANCH).

Income and Expenditure Account for the Year Ended December 31, 1959.

	£	s.	d.
To Secretarial Fees	400	0	0
„ Printing and Stationery	115	7	9
„ Postages and Duty Stamps	146	17	7
„ Code Address	3	3	0
„ Refund: Subscription	8	8	0
„ Capitation Fees:			
Southern Subdivision	111	15	0
Northern Subdivision	83	5	0
Federal Council	325	0	0
Australasian Medical Publishing Co. Ltd.	260	0	0
London Office	399	19	0
„ Wreaths	4	5	6
„ Audit Fees	21	0	0
„ Address to His Excellency the Governor	14	1	3
„ Bank Exchange	1	14	9
„ Cheque Book	3	0	0
„ Travelling Expenses	318	15	0
	2,216	11	10
Excess Income over Expenditure	437	5	10
	£2,653	17	8

	£	s.	d.	£	s.	d.	£	s.	d.
To Members' Subscriptions				3,444	1	0			
Less:									
Building Fund	459	10	0						
Post-Graduate Committee	115	0	0						
Library Fund (Southern Subdivision)	382	4	0						
				956	14	0			
							2,487	7	0
„ Interest:									
Commonwealth Bonds							6	10	9
Australasian Medical Publishing Co. Ltd.							72	12	5
E.S. & A. Bank Ltd.							2	6	6
„ Exchange								4	0
„ Sale Car Badges								17	6
„ Profit on Realization of War Savings Certificates								54	17	3
„ Profit Sale of Commonwealth Treasury Bonds								29	2	3
							£2,653	17	8

Balance Sheet as at December 31, 1959.

	£	s.	d.	£	s.	d.
Bank of New South Wales				4,580	3	3
Sundry Creditors:						
Post-Graduate Fund	17	10	0			
Library Fund	8	8	0			
Southern Subdivision	27	15	0			
Northern Subdivision	37	5	0			
				90	18	0
War Relief Contribution				2	11	0
Surplus *re* Dinner				32	11	6
Accumulated Fund Account—						
Balance January 1, 1959	3,087	12	0			
Plus Australasian Medical Publishing Co. Ltd.	129	19	11			
	3,217	11	11			
Surplus for Year	437	5	10			
				3,654	17	9
				£8,361	1	6

	£	s.	d.	£	s.	d.
Australasian Medical Publishing Co. Ltd.:						
Debentures	1,295	0	0			
Cash in Hand	7	6	2			
				1,302	6	2
Commonwealth Bonds				260	0	0
Furniture				30	0	0
Gestetner				95	0	0
Cash at E.S. and A. Bank				88	10	6
Mortgage Investment				500	0	0
Building Fund Account				6,085	4	10
				£8,361	1	6

Building Fund as at December 31, 1959.

	£	s.	d.
To Purchase Property Davey Street	9,000	0	0
„ Valuation Fees	13	13	0
„ Legal Fees	196	5	4
„ Architects' Fees	25	0	0
„ Purchases: Electric Stove	82	9	0
„ Plumbing Repairs	83	13	0
„ Bank Interest	139	12	0
„ Purchases: Blinds, etc.	60	0	0
„ City Rates	46	19	3
„ Insurance	8	6	3
	£9,655	17	10
To Balance January 1, 1960	£6,085	4	10

	£	s.	d.
By Balance January 1, 1959	1,196	13	4
„ Interest, Hobart Savings Bank	6	17	5
„ Subscriptions—Members	459	10	0
„ Rents Received	570	6	3
„ Donations—Members	1,302	6	0
„ Mortgage Interest	35	0	0
„ Balance, December 31, 1959	6,085	4	10
	£9,655	17	10

Audited and found correct, subject to our letter of February 26, 1960.

ADAMS AND BENETTO, Chartered Accounts (Aust.).

Honorary Medical Secretary: Dr. K. M. Kelly.

Branch Councillors: Dr. D. B. Nathan, Dr. J. W. Hunn, Dr. A. J. M. Dobson, Dr. K. S. Millingen, Dr. A. L. Stephenson.

ELECTION OF AUDITORS.

Messrs. Adams and Benetto were reelected auditors for 1960.

INDUCTION OF PRESIDENT.

Dr. L. H. Wilson introduced the incoming President, Dr. R. A. Lewis, and vacated the chair in his favour. Dr. Lewis thanked the members for his election.

REPORT ON THE ROAD SAFETY COUNCIL OF TASMANIA.

After a bewildering succession of Cabinet changes, Tasmania has, at last, a fairly permanent Minister of Transport, the Honorable J. B. Conolly, M.L.C. The new Minister made a visit to New Zealand, a country which is far in advance of Australia in matters of road safety. As a result of this visit, Cabinet action may be anticipated along these three lines: (*a*) compulsory safety helmets for motor cyclists; (*b*) speed limits; (*c*) compulsory inspection of motor vehicles.

It may be wondered why similar action was not initiated by the Road Safety Council of Tasmania. Indeed these matters were discussed. The Council, which is advisory only, is largely composed of representatives from the numerous and varied branches of the motor trade. These representatives show a marked reluctance to the making of any change.

F. PHILLIPS.

WORKERS' COMPENSATION SUBCOMMITTEE.

Since the last annual report, an approach has been made on several occasions to the Fire and Underwriters' Association and to the Chief Secretary to have: (*a*) the fee schedule for visits and consultations brought into line with current general practitioners' fees, because it is considered that a concessional service is no longer warranted; (*b*) a schedule of fees for operations performed by specialist surgeons as is current in New South Wales at the request of the State Committee of the Royal Australasian College of Surgeons; (*c*) variations in the radiology fees to bring them into line with present-day techniques and charges. The negotiations were prolonged and sometimes acrimonious, and the final result will be that on February 3, 1960, a compromise which has been reached will operate on item (*a*), but the other two objectives have been rejected.

As a result, the rate for surgery and hospital consultations and visits has risen by 2s. 6d. making the fee for first visit at surgery 17s. 6d. and at hospital £1. The remaining parts of the schedule are unchanged.

It may be necessary to take further action in the coming year.

FRANKLIN R. FAY.

RETIRING PRESIDENT'S ADDRESS.

Dr. L. H. Wilson then delivered his retiring president's address (see page 953).

Out of the Past.

HONOURS TO MEDICAL MEN[1].

[From the *Australasian Medical Gazette*, May, 1901].

THE list of honours of medical men who have served with the Australian contingents in South Africa have been published. We heartily congratulate our colleagues who have thus gained distinction on the battle field: at the same time we must confess to some degree of disappointment at the number and quality of the decorations. We have become so accustomed to seeing the excellence of the New South Wales Army Medical Corps proclaimed by commanding officers and war correspondents alike, that we probably expected too much.

Colonel Williams to whom is due the whole of the credit of bringing the New South Wales Army Medical Corps to its great state of efficiency, and who was entrusted with an important appointment in South Africa, has been made a Companion of the Bath (C.B.): we certainly could have wished for a knighthood for him. Majors Fiaschi, Roth and Perkins and Captain T. A. Green have been made Companions of the Distinguished Service Order (D.S.O.): we are sure they all thoroughly won their decorations. Several of the medical officers of the Corps were promoted in rank during the campaign, as from Captain to Major, or from Lieutenant to Captain, and we understand that at least two or three of those who received no decorations were specially mentioned by their Commanding Officers. The nursing sisters who left Sydney with the Second Contingent, have also come in for high commendation from Lord Roberts.

The profession in Victoria has been honoured in the person of Sir Thomas Fitzgerald, who has been made a K.C.B. for a period of honorary service in South Africa. We heartily congratulate Sir Thomas on his additional honour.[2]

[1] From the original in the Mitchell Library, Sydney.

[2] Amongst subsequent honours Captain N. R. Howse was awarded the Victoria Cross, Major W. L. Eames the C.B. and Lieutenant A. H. Horsfall the D.S.O.

Special Correspondence.

LONDON LETTER.

BY OUR SPECIAL CORRESPONDENT.

National Census.

THE sixteenth census of the population of Great Britain will take place on Sunday, April 23, 1961. The first census was in 1801, and it has been continued at ten-year intervals, except for the year 1941.

Information will be required for every person alive in the country at midnight ending the census day and will include age, sex, birthplace, nationality, address, marital condition, date of marriage and number of children of married women, the type of dwelling and household amenities. New questions introduced for the first time concern the qualifications of scientists and technologists and seek some guide to the movement of population within the country.

Improving Efficiency in Hospitals.

A year ago the Minister of Health appointed a committee to advise on measures for improving efficiency in the National Health Service in England and Wales. This Advisory Council consists of 14 members, representatives from medicine, nursing, hospital and social administration, trade unions and industrial management. It has no executive powers but will seek by persuasion, education and training to stimulate initiative and a progressive attitude of mind in the field of management efficiency on the part of all concerned in the Health Service.

The professional aspects of clinical practice will not be the main concern of the Council, but by improving the general organization of hospital work, it seeks to back up the progress expected from the increasing advances of science and technology. Progress will inevitably bring changes in attitude, in methods and in organization. The Council will try to foster a climate of opinion throughout the Service in which better methods will be not only accepted but welcomed.

Hospital Meals.

As a result of the findings of a pilot survey of the catering conditions of eight hospitals in Cornwall, the Nuffield Provincial Hospitals Trust has provided a grant of £27,000 for a comprehensive investigation of hospital meals on a national scale. The pilot survey was carried out by a team from the London School of Hygiene and Tropical Medicine under the direction of Professor B. S. Platt of the Department of Nutrition. The results showed that about half the meals served were inadequate from a nutritional standpoint—lacking in protein balance and with excessive loss of vitamin C in vegetables. There was apparent lack of relationship between the nutritional qualities of the food and the illnesses for which the patient was being treated. Because of impalatability, more often than not, only half the food was eaten.

There are many difficulties in cooking and serving food for hundreds of people at one time, and it was considered doubtful if the best of existing hospital arrangements would ever be entirely satisfactory. Skill and knowledge

should be concentrated in centres where, with modern equipment and techniques, meals can be prepared, packaged and held in deep-freeze until immediately before being served to the patient.

The National Survey is expected to take up to three years, and the investigators will sometimes visit hospitals unannounced to see clearly hospital catering as it really is.

Birth Control.

In the fourth Reith Lecture on the B.B.C. Home Service, on "The Future of Man", Professor Medawar of University College, London, declared that there was no reason to believe that world-wide adoption of birth-control would have biologically malign effects. On the contrary, failure to adopt family limitation would lead, in the long run, to misery, privation and economic distress. What people really feared when they talked of the biological evils of birth control was the theoretical possibility that, in terms of tens or hundreds of generations, the proportion of innately very fertile men and women might go down. It was a fallacy to assume that the fertility of a species was a kind of primeval fixture, as if some animals and plants were driven by some demon of fertility to have vastly more offspring than were needed. It is sometimes said that the explanation of natural selection itself is that living things produce an allegedly "prodigious" number of offspring, of which only a chosen few are spared. But to say this is to forget that the level of fertility developed by any species is just as much the consequence of natural selection as its cause. In fact, there is no good reason to fear that an innate decline in fertility must be a stage on the road to extinction or that we should face a struggle to keep mankind alive.

At a conference in London of the International Planned Parenthood Association, the progress of experimental trials of oral contraceptives in Britain was reported by the experts present. Dr. Margaret Jackson, a leading authority on fertility in women, spoke of her experience in tests of the progesterone compounds "Enavid" and "Primolut N". It appeared that the results of her experiments with women volunteers in Devon and London were possibly 100% effective, but side-effects tended to make the drugs unpopular; until this was overcome, these methods would not be universally acceptable. Reduction of dose was important, and experiments with gradual reduction of dose were being tried. Dr. Eleanor Mears, Secretary of the Family Planning Association, declared that the women's fertility had not been impaired. All the experts at the conference were very chary of claiming proven success in any of these trials, and there was general agreement that the long-term effects were not yet known.

Drug Addiction.

Two years ago, the Minister of Health appointed a committee, under the chairmanship of Sir Russell Brain, to advise him on drug addiction. An interim report has now been published, which relates particularly to the drugs carbromal and bromvaletone, and to the extent to which the gases used by anæsthetists result in addiction. Carbromal and bromvaleton are two examples of drugs now on sale without restraint. They have been extensively advertised with the claim that they will relieve mild but widespread nervous disorders. It is clear that they have been widely used by the public, largely without medical guidance. The committee recommends in general that any drug or pharmaceutical preparation which has an action on the central nervous system and is liable to produce physical or psychological deterioration should be confined to supply on prescription.

Anæsthetic Gases.

In 1959 a consultant anæsthetist was found guilty of manslaughter in the case of the death of a patient under anæsthesia for a surgical operation. In the course of the proceedings it was stated that this anæsthetist had been addicted for some years to anæsthetic gases.

An accurate estimate of the incidence of addiction among anæsthetists is difficult to determine. Twenty cases have come to notice in the past 11 years. This incidence is very small if we consider the number of anæsthetics given, but in two known instances the patient's life has been endangered. It appears to the committee that, with the apparatus at present in use, the preliminary sniffing of the gases immediately before administration is a recognized and indispensable practice, and neglect of this measure may amount to professional negligence. To the great majority of anæsthetists, this practice offers no encouragement to addiction. The committee is convinced that anyone who is addicted to the inhalation of anæsthetic gases should not be entrusted with their administration, and the responsibility, in the first instance, for dealing with this irregularity lies with the anæsthetist's professional colleagues.

Correspondence.

AN APPEAL: FREDERIC WOOD JONES.

SIR: There is listed in the bibliography of Frederic Wood Jones (published at the end of his obituary notice in the *Journal of Anatomy*) the following reference:

> 1932 "The Island of Tragedy and Romance (Pearson Island)" Table Talk. Christmas Annual, 12-13.

Neither the Public Library of Victoria nor the *Herald* Library has been able to trace this Annual. Mr. Keith Manzie, who was associated with *Table Talk* during these years, assures me that there was such a publication. If anybody could lend me this particular Christmas Annual of 1932, I would be extremely grateful.

Yours, etc.,

BARRY E. CHRISTOPHERS.

366 Church Street,
Richmond, Victoria.
May 26, 1960.

THE MANAGEMENT OF EARLY BREAST CANCER.

SIR: The paper by Ackland, Holman and Stoll on the above subject is on a difficult and controversial subject. It sets out one set of opinions, and while one may disagree, it is their prerogative to hold these and make a clinical trial of the method advocated. Exception must be taken, however, to gross misrepresentations of another author's work.

Ovarian ablation has played no part in the treatment of breast cancer *ab initio* as described by Professor McWhirter. Indeed, not a few of his patients have borne children subsequent to their treatment. It is therefore invidious to ascribe his excellent results in a completely unselected large series to the palliation received from alteration in hormonal levels. No figures are given in the article, but for comparison Haagensen's five-year survival rate reported in 1951 was 48·7%. These patients were highly selected by biopsy of the internal mammary chain and the apex of the axilla before radical mastectomy was embarked upon. The purpose of these manœuvres is to select Stage II cases for operation, and in addition certain limits are placed upon the size of the primary, the size of the axillary nodes and the condition of the skin. McWhirter's figure for all cases including 569 Stage IV cases was 42%. When the "operable group"—i.e., clinical Stages I and II—are considered, 58% survived five years. Restricting the size of the primary improves the figure even further. It is emphasized that, as Mr. Ackland says, 15% of the Stage I and 58% of the Stage II will have undetected internal mammary metastases, and the supraclavicular nodes will be involved, although not palpably, in a small percentage of Stage I and 33% of Stage II.[2] These cases would have been deemed inoperable by Haagensen. Does Mr. Ackland consider the operation carried out by Haagensen to be indifferently performed?

McWhirter's condemnation of radical mastectomy with or without radiotherapy was not based solely on the poor results obtained. In passing, the "certain hospital" concerned was the Royal Infirmary of Edinburgh during Sir John Fraser's heyday. The change to simple mastectomy was made partly on logical grounds, and partly because it was realized that the results of a form of treatment should be related to all the patients presenting with the disease and not solely to those operated on. Any treatment for almost any disease not immediately fatal can be made to show good results by selecting those cases "most suitable" for the therapy. The remainder of the patients, even although they constitute, as in breast carcinoma, 64% of the total presenting, are offered only palliative treatment. Surely, Sir, any policy which is applicable to only one-third of the patients cannot be called a routine. One could not in all conscience say to patients: "I can offer 36% of you a chance of cure, although there is a treatment which offers 85% of you a chance of cure. If you are in the fortunate

one-third to whom both treatments are applicable, the chances of cure are approximately the same, and I have no method of knowing until your axilla is opened and you are committed to the first policy whether, in fact, you are one of the fortunate third."

Certainly the patient loses her breast in both forms of therapy; but in only one does she lose her pectoralis major, suffer increasing fibrosis of her axilla and run a major risk of having a stiff shoulder and an œdematous arm. The statement that the morbidity of both operations is practically the same is simply not true.

The same applies to "radiaton cannot destroy axillary metastases with any certainty", depending upon the interpretation of the phrase "any certainty". As far as I know, no treatment of any malignant disease, and few treatments of any other conditions, can be offered to a patient with a 100% five-year guarantee of success. But if radiation can, as McWhirter's results show, destroy metastases in the supraclavicular and internal mammary regions—and indeed, in the present technique advocated, radiation is relied upon to do just that—why not treat all the lymph-drainage areas the same way? The increase in the size of the fields to cover the whole axilla adequately, particularly with supervoltage, will not necessitate any reduction in dose levels. A further question is, what happens at the point where surgery and X-ray therapy meet? If overlapping occurs, as it must, that area of the axilla will receive full X-ray therapy on top of devitalized tissue, thus increasing the fibrosis which is already present and treating tumour cells rendered radio-resistant by the surgical interference.

My intention is solely to correct false statements made about the work of a rival in the same field—one who has shaken "the firm establishment" of radical mastectomy as the treatment of choice in early breast carcinoma. To misquote his writings and omit any reference at the end of the article suggests that his work has not been consulted in the original, although one of McWhirter's articles lies cheek by jowl with one of the other references.

The remainder of the article is adequately answered in McWhirter's publications to which I refer all those interested in the treatment of breast carcinoma.

The controversy will of course go on; but no one has yet refuted the logic of the Edinburgh technique, and no one has published better results in a totally unselected series of cases.

The greatest good for the greatest number?

Yours, etc.,

65 Union Road,
Surrey Hills,
Victoria.
Undated.

T. F. SANDEMAN.

References.

HAAGENSEN, C. D., and STOUT, A. P. (1951), "Carcinoma of the Breast. Results of Treatment", *Ann. Surg.*, 134:151.

MCWHIRTER, R. (1949), "Cancer of the Breast", *Amer. J. Roentgenol.*, 62:335.

MCWHIRTER, R. (1955), "Simple Mastectomy and Radiotherapy in the Treatment of Breast Cancer", *Brit. J. Radiol.*, 28:128.

MCWHIRTER, R. (1957), "Some Factors Influencing Prognosis in Breast Cancer", *J. Fac. Radiol.*, 8:220.

SIR: In your issue of May 21, 1960, you published an article by Ackland, Holman and Stoll on "The Management of Early Breast Carcinoma". This is a most disappointing contribution to the problem under review. Many of the declamations contained therein are highly debatable, and no supportive data (based on local Victorian experience) are adduced for consideration. In view of this, the presentation surely should have been couched as an expression of opinion and not as a dogmatic tenet. I quote paragraph 6 of their summary: ". . . . simple mastectomy has no place in the treatment of potentially curable carcinoma . . . ."

To give value to their opinions, or even perspective to their views, they should have indicated the extent of their own experience with simple mastectomies. Comparison of the two methods of treatment is already a matter if international controversy, and the presentation of an unsubstantiated statement of opinion, such as this, does little to elucidate the problem, which is already clouded by prejudice and emotion on both sides. Even in an assessment of the radical mastectomy, where their experience is no doubt unassailable, their plea for stricter selection does not indicate the fate of the 70% or 80% of cases excluded by this approach; nor does the consequent "improved" five-year figure give any indication of the degree of selection implied, far less the impact of the restricted treatment on the whole problem of breast cancer.

Yours, etc.,

KEITH S. MOWATT,
Deputy Director.

The Queensland Radium Institute,
Brisbane.
May 26, 1960.

GENERAL PHARMACEUTICAL BENEFITS.

SIR: An old *Punch* cartoon depicts the maid's room in the attic.

Visitor (seeing the water dripping through the ceiling): "Isn't it rather damp?"

Mistress: "Not for a servant."

I was reminded of this when I read of the phenylbutazone and the pensioner affair (*Hansard*, March 22, 1960; MED. J. AUST., May 21, 1960). Did the following sort of dialogue take place with the Pharmaceutical Benefits Advisory Committee?

Expert A: "Isn't 'Butazolidin' rather toxic?"

Expert B: "Not for a pensioner."

Yours, etc.,

J. E. REES.

The Talbot Clinic,
Rockhampton, Queensland.
June 1, 1960.

SIR: I wish to express my complete disagreement with this odious scheme, which is undue interference in the doctor-patient relationship, and tantamount to civil conscription. On these terms it should be fought to its final rejection and defeat. C. R. Watson's (MED. J. AUST., May 28, 1960) idea is an excellent one, and I have carried out his suggestion forthwith.

Our universities and teachers should also be approached to express their views on this very serious problem. I cannot suggest anything better than hold a general meeting of all general practitioners of this opinion and that we burn these drug books in public, very much the same way as the coloured human beings burn their passes in South Africa.

Yours, etc.,

R. TRAUER.

52 Fitzroy Street,
St. Kilda,
Victoria.
May 31, 1960.

"FINAL NOTICE."

SIR: Dr. L. J. Shortland's letter (MED. J. AUST., May 28, 1960) headed "Final Notice" was discussed at a recent meeting of the Council of the Medical Defence Union, and it was resolved to answer his comments.

The New South Wales Medical Defence Union is not an insurance company or financial institution, but a company formed by members of the profession for protection in medico-legal matters. The members of its Council receive no payments for their work, which is often tedious and difficult, and with one exception, they are men with at least thirty years of experience in practice. The Union provides protection, assistance and advice far in excess of that available under any medical indemnity policy.

To provide this cover, it is necessary to reinsure and to pay a premium in respect of every member whose name remains on the books, whether he is financial or unfinancial. Although an unfinancial member automatically forfeits the privileges of protection in respect of occurrences while he is unfinancial, he is still covered for a period of six years in respect of occurrences during his financial membership, subject to resumption of active membership in accordance with the Articles.

The Articles of Association clearly set out a member's responsibility in regard to payment of subscriptions. A notice is posted in January of each year, pointing out that subscriptions are due on or before March 1. (The financial year ends on February 28.) If the member fails to pay by this date, a reminder notice is sent out early in March, and if the subscription is still unpaid at March 31, a final notice is sent notifying the member that he is unfinancial.

Thus a member has thirty-one days' grace before being posted as unfinancial, and not seven, as suggested by Dr. Shortland.

Dr. Shortland appears to suggest that an unfinancial member should be allowed to pay a proportionate amount for the balance of the financial year on resuming membership. This could be considered to be unfair by his fellow members, and it could mean that the Union could pay more in reinsurance than was received in subscription from the member concerned.

It will be appreciated that the rules of the Union have been formulated to meet the requirements of the reinsurers and to safeguard the members as a whole.

Yours, etc.,

S. H. LOVELL,
President.

N.S.W. Medical Defence Union Limited,
135 Macquarie Street,
Sydney.
June 7, 1960.

Post-Graduate Work.

THE MELBOURNE MEDICAL POST-GRADUATE COMMITTEE.

PROGRAMME FOR JULY, 1960.

THE Melbourne Medical Post-Graduate Committee announces the following programme for July, 1960.

Pathology for Primary F.R.A.C.S.

A short course in the principles of pathology, suitable for candidates for the Primary F.R.A.C.S., will commence on Monday, July 4, at the University of Melbourne Pathology Department, at 1.30 p.m., and continue for 12 weeks. The fee for this course is £7 7s., and enrolments should be received by June 20. (Anatomy classes will commence at 2.45 p.m. from July 4.)

Neuropathology.

The Committee will conduct a short course of five or six lectures by Dr. Ross Anderson on Mondays, at 7.30 p.m., at the Alfred Hospital Pathology Department. This will commence on July 4. The fee is £5 5s., and entries close on June 23.

Course in Anæsthetics.

The Victorian State Committee of the Faculty of Anæsthetists is holding a full-time two weeks' course of lectures, at the Royal Australasian College of Surgeons, Spring Street, Melbourne, from July 4 to 15. The subscription of £10 10s. is payable to Mr. H. G. Wheeler, Secretary of the R.A.C.S. Trust Account, at the above address. Further inquiries should be directed to Dr. H. P. Penn, at JA 1334, or to Dr. H. D. O'Brien at JA 6568, or both at St. Vincent's Hospital, Melbourne.

Overseas Visitors.

Professor S. Leon Israel, Professor of Obstetrics and Gynæcology in the University of Pennsylvania, will visit the University of Melbourne from July 23 to 30. During this week the following programme has been arranged at the Royal Women's Hospital: Tuesday, July 26, at 1 p.m., fifth floor—obstetrical ward round; Thursday, July 28, at 1.15 p.m., sixth floor—obstetrical discussion; Friday, July 29, at 4 p.m., sixth floor—gynæcological discussion. These will be open to general practitioners.

Professor J. Goligher, F.R.C.S., of the Department of Surgery, University of Leeds, will be Guest Professor at the Alfred Hospital for one month from July 18.

PRELIMINARY ANNOUNCEMENTS.

Pædiatric Refresher Week.

A pædiatric refresher week will be held at the Royal Children's Hospital, Melbourne, from Monday, August 29, to Friday, September 2, inclusive. This will be conducted by the honorary staff of the hospital; it will begin each day at 9.30 a.m. and continue till 4 p.m. Sessions will include symposia on rheumatic fever, cerebral palsy and neonatal problems, as well as discussions of subjects such as differentiation of acquired from congenital heart disease, infective diarrhœa, common poisonings, cœliac disease, the current status of therapy in leukæmia and malignant disease in childhood, appendicitis, osteomyelitis, testicular conditions, management of cleft lip and palate and fracture of the upper limb. Neonatal and cardiac quiz sessions, clinical meetings and cardiac and dermatology demonstrations will also be part of the programme.

The enrolment fee is £2 2s., and enrolment should be made with the Melbourne Medical Post-Graduate Committee on their special form, by August 15.

Academic Programme at St. Vincent's Hospital.

In October, 1960, an academic programme, extending over one month, will be presented at St. Vincent's Hospital to mark the fiftieth anniversary of the establishment of the hospital as a clinical school and the opening of the new wing. Professor Claude E. Welch, Professor of Surgery, Harvard Medical School, will be the guest surgeon during this time, and will conduct operating and teaching sessions. The Committee's subscribers are invited to attend. A full programme will be published later.

RECORDED LECTURES.

The following recording has been added to the Committee's library of 10 in. microgroove discs, with accompanying illustrations on 2 in. by 2 in. slides, and may be borrowed without charge: "Cancer of the Stomach", a symposium conducted by the Melbourne Medical Post-Graduate Committee; nine short lectures by Professor R. D. Wright, Dr. A. V. Jackson, Dr. R. R. Andrew, Dr. W. Hare, Dr. W. E. King, Dr. L. E. Hurley, Sir William Upjohn, Mr. Grayton Brown and Dr. E. V. Keogh, delivered on April 2; discs run for two and a half hours, 75 slides.

ADDRESS.

The address of the Melbourne Medical Post-Graduate Committee is 394 Albert Street, East Melbourne. Telephone: FB 2547.

THE POST-GRADUATE COMMITTEE IN MEDICINE. IN THE UNIVERSITY OF SYDNEY.

PROGRAMME FOR JULY, 1960.

THE Post-Graduate Committee in Medicine in the University of Sydney announces the following courses, to be held during the month of July, 1960.

Post-Graduate Training in Cardio-Vascular Diseases.

The third of the series of evening seminars on various aspects of cardio-vascular diseases suitable both for general practitioners and for post-graduates studying for higher degrees or diplomas will deal with heart disease in younger people. These seminars, which will be under the supervision of Dr. G. E. Bauer, will be held in the Maitland Lecture Hall, Sydney Hospital, and the programme will be as follows:

Wednesday, July 6: 8 p.m., symposium on "Rheumatic Fever—Pathology, Pathogenesis, Diagnosis and Treatment", Professor Lorimer Dods, Dr. Bryan Dowd, Dr. R. D. K. Reye, Dr. Helen Walsh; 9.30 p.m., panel discussion.

Wednesday, July 13: symposium on "Valvular Heart Disease": 8 p.m., "Mitral Stenosis", Professor R. B. Blacket, Dr. R. G. Epps; 8.45 p.m., "Aortic Stenosis", Dr. G. E. Bauer, Dr. H. P. B. Harvey; 9.30 p.m., panel discussion.

Wednesday, July 20: 8 p.m., symposium on "Congenital Heart Disease": medical aspects, Dr. J. F. Farrar, Dr. W. A. Seldon, Dr. D. S. Stuckey; surgical aspects, Mr. Rowan Nicks; 9.30 p.m., panel discussion.

Wednesday, July 27: 8 p.m., "Pericarditis" (lecturer to be confirmed); 8.30 p.m., "Subacute Bacterial Endocarditis", Dr. J. B. Hickie; 9 p.m., "Cardiomyopathy", Dr. J. G. Richards; 9.30 p.m., panel discussion.

The inclusive fee for attendance is £2 2s.

Week-End Course in Endocrinology.

A week-end course in endocrinology suitable for general practitioners will be held on July 16 and 17 in the Scot Skirving Lecture Theatre of the Royal Prince Alfred Hospital. The course will be under the supervision of Dr. K. S. Harrison, and the programme will be as follows:

Saturday, July 16: 10.15 a.m., introduction; 10.30 a.m., "Endocrine Variations of Puberty", Dr. R. H. Vines; 11.15 a.m., "Primary Amenorrhœa as an Endocrine Problem", Associate Professor R. P. Shearman; 12.15 p.m., "Para-

thyroid Disorders", Dr. M. R. Playoust; 2 p.m., "Investigation of Adrenal Disorders", Dr. J. M. Greenaway; 2.45 p.m., "Hyperfunction of the Adrenal Cortex", Dr. K. S. Harrison; 4 p.m., panel discussion—"Adrenal, Gonadal and Pituitary Disorders'.

Sunday, July 17: 9.30 a.m., "Assessment of Thyroid Function", Dr. I. D. Thomas; 10.15 a.m., "Treatment of Hyperthyroidism", Professor F. F. Rundle; 11.30 a.m., panel discussion—"Thyroid and Parathyroid Disorders".

The fee for attendance is £3 3s.

Week-End Course in Orthopædics.

A week-end course in orthopædics will be held on July 23 and 24 in the Students' Lecture Room, Royal North Shore Hospital. The course will be under the supervision of Dr. C. D. Langdon, and the programme is as follows:

Saturday, July 23: 2 p.m., "Osteomyelitis — the Present Position', Dr. K. Daymond; 2.20 p.m., "Club Foot and Congenital Dislocation of the Hip", Dr. B. C. Bracken; 2.40 p.m., "Elbow Injuries", Dr. R. Middleton; 3.15 p.m., "Growth Deformities", Dr. R. McGlynn; 3.35 p.m., "Shoulder and Arm Pain', Dr. C. D. Langton; 3.55 p.m., film.

Sunday, July 24: 10 a.m., "Osteoarthritis—Conservative and Surgical Management", Professor B. McFarland, F.R.C.S. (England), F.R.C.S. (Edinburgh), Professor of Orthopædic Surgery, University of Liverpool, and Official Overseas Lecturer of the Australian Post-Graduate Federation in Medicine for 1960; 11 a.m., "The Great Toe Joint", Dr. A. R. Hamilton; 11.45 a.m., "Compound Fractures", Dr. C. D. Langton; 12.15 p.m., "Backache', Dr. R. McGlynn; 2 p.m., "Footwear, Insoles and Splints. The Thomas Splint", demonstration, Dr. R. Middleton; 2.45 p.m., "Fractures of the Femur and Tibial Shaft", Dr. B. C. Bracken; 3.30 p.m., "Cut Tendons and Nerves', Dr. A. Parker.

The fee for attendance is £3 3s.

Course in Clinical Respiratory Physiology.

A course in clinical respiratory physiology will be conducted in the Department of Medicine, University of Sydney, from Tuesday, July 12, to Thursday, August 11, 1960. The course, which will be under the supervision of the Professor of Medicine, Professor C. R. B. Blackburn, will consist of 10 one-hour sessions of a lecture-demonstration-discussion nature. Each session will begin at 4 p.m., and the programme is as follows:

Tuesday, July 12: introduction, Dr. John Read; "Some Physical Principles Involved in Respiration", Dr. K. T. Fowler.

Thursday, July 14: "Oxygen and Carbon Dioxide in Gas and Blood. pH". Dr. John Read and Dr. H. J. H. Colebatch.

Tuesday, July 19: "Ventilation of the Lungs", Dr. John Read.

Thursday, July 21: "Ventilation—Blood Flow Relationship Within the Lungs", Dr. J. Read.

Tuesday, July 26: "Pulmonary Circulation", Dr. D. F. J. Halmagyi.

Thursday, July 28: "Diffusion", Dr. John Read and Dr. H. J. H. Colebatch.

Tuesday, August 2: "Mechanics of Breathing", Dr. H. J. H. Colebatch.

Wednesday, August 3: "Control of Respiration, Anoxia", Professor P. I. Korner.

Tuesday, August 9, and Thursday, August 11: discussion of individual patients and disease states; therapeutic principles; question and answer session—Dr. John Read, Dr. K. T. Fowler, Dr. D. F. J. Halmagyi, Dr. H. J. H. Colebatch.

The fee for attendance is £5 5s.

Course on the Management of Cardiac Arrest.

A one-day course in the management of cardiac arrest will be held, under the supervision of Dr. Bruce Clifton, in the Page Chest Pavilion, Royal Prince Alfred Hospital, and the Department of Surgery, University of Sydney, on Friday, July 8, 1960. The programme is as follows: 9 a.m., welcome and introduction, Professor John Loewenthal; to be followed by "General Aspects About the Problem of Cardio-vascular Collapse in Surgical Practice", Dr. Bruce Clifton; 11 a.m., "Prevention of Cardio-vascular Collapse", Dr. W. I. T. Hotten; 11.30 a.m., general discussion; 1.15 p.m., "Practical Aspects of Cardiac Resuscitation", Mr. Rowan Nicks; 1.45 p.m., practical demonstration and instruction in the animal operating theatre, Mr. Rowan Nicks, Dr. Bruce Clifton (visitors to participate).

The course will be limited to 10 members, and the fee for attendance £4 4s.

Method of Enrolment.

Those wishing to enrol in any of the foregoing courses are requested to make early written application, enclosing remittance, to the Course Secretary, the Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephone: BU 4497-8. Telegraphic Address: "Postgrad Sydney".

ANNUAL SUBSCRIPTION COURSE.

The Post-Graduate Committee in Medicine in the University of Sydney announces the following programmes for overseas lecturers expected shortly.

Dr. S. Leon Israel, Professor of Gynæcology and Obstetrics, University of Pennsylvania, will give the following lectures: Monday, June 27, 2 p.m., Maitland Lecture Hall, Sydney Hospital: *"Mittelschmerz,* Cyclic Mastalgia and Premenstrual Tension"; Tuesday, June 28, 2.15 p.m., Royal Hospital for Women: "Sterility"; Wednesday, June 29, 11.45 a.m., St. Vincent's Hospital: "Pre-Menopausal and Post-Menopausal Abnormal Uterine Bleeding'; Wednesday, June 29, 8.15 p.m., Stawell Hall, 145 Macquarie Street: "Dysmenorrhœa"; Thursday, June 30, 11 a.m., King George V Memorial Hospital: "Amenorrhœa of Hypothalamic Origin, Amenorrhœa of Pituitary Origin, Amenorrhœa of Ovarian Origin"; Tuesday, July 5, 8.15 p.m., I.C.I. Theatrette, I.C.I. House, 69 Macquarie Street: "Amenorrhœa, or Menstrual Disorders of Puberty"; Wednesday, July 6, 2 p.m., The Royal North Shore Hospital: "Dysmenorrhœa"; Thursday, July 7, 2 p.m., The Women's Hospital, Crown Street, Lecture Hall: "Recurrent (Habitual, Repeated) Abortion"; Tuesday, July 12, 8 p.m., St. George Hospital: "Amenorrhœa of Hypothalamic Origin".

Professor James Dauphinee, Professor of Pathological Chemistry, University of Toronto, will give the following lectures: Wednesday, July 6, 2 p.m., Maitland Lecture Hall, Sydney Hospital: "Laennec's Cirrhosis — An Analysis of Ætiology, Clinical Manifestations and Prognosis in 150 Patients"; Wednesday, July 6, 8.15 p.m., Stawell Hall, 145 Macquarie Street: "The Clinical Chemistry Laboratory in Medical Diagnosis"; Wednesday, July 13, 8 p.m., Robert H. Todd Memorial Hall, 135 Macquarie Street (in association with the Section of Pathology of the New South Wales Branch of the British Medical Association); "Paper and Starch Gel Electrophoresis in the Study of the Serum Proteins"; Tuesday, July 19, 8.15 p.m., Stawell Hall, 145 Macquarie Street (in association with The College of Pathologists of Australia): "Serum Enzymes and their Usefulness in Clinical Diagnosis"; Wednesday, July 20, 2 p.m., Students' Lecture Theatre, St. Vincent's Hospital: "Plasma Proteins in Health and Disease"; Thursday, July 21, 8.15 p.m., Stawell Hall, 145 Macquarie Street: "Water and Electrolyte Metabolism and Abnormalities in Human Disease".

Professor Bryan McFarland, Professor of Orthopædic Surgery, University of Liverpool, will visit Sydney from Saturday, July 16, to Friday, August 5, as the Category A Lecturer of the Australian Post-Graduate Federation in Medicine. Details of Professor McFarland's programme will be announced shortly.

The annual subscription course covers attendance at lectures by overseas lecturers and other specially arranged activities. The annual fee is £3 3s. from July 1. The fee for first-year and second-year resident medical officers is £1 12s. 6d. Last-minute alterations to meetings are notified by advertisement in *The Sydney Morning Herald* ("Public Notices"), if possible on the day before the meeting.

THE AUSTRALIAN POST-GRADUATE FEDERATION IN MEDICINE.

First Australian Conference on Post-Graduate Medical Education.

THE Australian Post-Graduate Federation in Medicine announces that the first Australian Conference on Post-Graduate Medical Education will be held at the School of Public Health and Tropical Medicine, University of Sydney, from Wednesday, August 10, to Friday, August 12, 1960.

The theme of the Conference will be "Australian Post-Graduate Medical Education during the First Five Years after Graduation". The objective of the Conference will be to survey the present opportunities for post-graduate medical education for Australians, to study the particular problems involved and to discuss the lines of future development. After the opening of the Conference, the Conference will split into four groups for single, combined and sub-group discussions. The Conference will conclude with panel discussions and plenary sessions to receive the reports of the chairmen ad rapporteurs of each group. The groups, their chairmen and officers are as follows:

Group I—Surgery, Gynæcology, Obstetrics and Allied Specialties: Chairman, Mr. I. B. Jose; Vice-Chairman, Mr. Douglas Miller; Secretary Rapporteur, Mr. P. J. Kenny; Secretary Rapporteur, Mr. S. F. Reid.

Group II—Medicine and Allied Specialties: Chairman, Professor J. G. Hayden; Vice-Chairman, Dr. K. B. Noad; Secretary Rapporteur, Dr. Selwyn Nelson; Secretary Rapporteur, Dr. T. H. Hurley.

Group III—General Practice: Chairman, Dr. C. C. Jungfer; Vice-Chairman, Dr. H. Stuart Patterson; Secretary Rapporteur, Dr. J. G. Radford; Secretary Rapporteur, Dr. H. M. Saxby.

Group IV—Academic Departments and Full-Time Laboratories: Chairman, Professor C. R. Bickerton Blackburn; Vice-Chairman, Professor R. W. Hawker; Secretary Rapporteur, Professor A. J. Day; Secretary Rapporteur, Dr. W. J. Hensley.

Attendance at the Conference is open to all organizations and persons interested in medical education. Accommodation at reasonable cost has been arranged at Wesley College, University of Sydney, and at the Royal Hospital for Women, for about 50 visitors. The registration fee is £1 1s. All sessions of the Conference will be recorded, and a report of the Conference will be published. The registration fee may be forwarded to the Honorary Secretary, Australian Post-Graduate Federation in Medicine, The University of Adelaide, North Terrace, Adelaide, or to the Honorary Director, Post-Graduate Committee in Medicine in the University of Sydney, 131 Macquarie Street, Sydney, from either of whom further details may be obtained.

Notes and News.

The First All-Asian Congress of Pædiatrics, New Delhi.

The first All-Asian Congress of Pædiatrics will be held at New Delhi, India, from January 2 to 6, 1961. The Congress is sponsored by the Indian Pediatric Society and the Association of Pediatricians, with the approval of the Government of India. Pædiatricians from all countries of geographic Asia are cordially invited to participate in the Congress, as individuals or as representatives of the national pædiatric societies or of the governments or universities. Pædiatricians from countries other than Asia may attend as observers. The official languages of the Congress will be Chinese, English, Hindi, Japanese and Russian. Registration of members must be notified on or before June 30, 1960. Titles of papers, exhibits, films, etc., with an abstract of not more than 200 words, should reach the Secretary-General on or before June 30, 1960. Abstracts of the papers will be considered by the Scientific Programme Committee, and papers accepted for inclusion in the programme will be communicated to the participants by June 30, 1960. Delegation fees must be paid in rupees, dollars or sterling, and cheques or draft must be drawn in favour of "First All-Asian Congress of Pædiatrics" and crossed. The fees are as follows: regular members or observers, £4 4s. sterling; associate members, £3 3s. sterling; student members, £1 10s. sterling. Registration forms will be available on request from the Secretary-General's Office. Further information may be obtained by writing to The Secretary General, The First All-Asian Congress of Pædiatrics, The Institute of Child Health, 95, Dilkhusa Street, Calcutta 17, India.

Imported Toys Held in Quarantine.

The Commonwealth Department of Health has impounded thousands of imported plastic toys found to contain soil or prohibited seeds. The importers have been informed that the toys must be destroyed or returned to the exporting country in Asia. The Commonwealth Minister for Health, Dr. D. A. Cameron, has stated that this action was a necessary safeguard against human, animal and plant diseases entering Australia and spreading within

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED MAY 14, 1960.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	..	3(2)	1	..	1(1)	..	..	..	5
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	4	..	3	..	..	..	9	..	16
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	4(2)	8(7)	1(1)	..	6(3)	..	3	..	22
Diphtheria	1	..	..	..	..	..	..	..	1
Dysentery (Bacillary)	..	..	..	1	2(2)	..	..	..	3
Encephalitis	..	..	..	..	..	..	..	..	..
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	53(23)	31(15)	19(2)	8(8)	2(2)	..	..	..	113
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	4	..	..	..	..	..	4
Malaria	..	1(1)	2(2)	..	..	..	..	..	3
Meningococcal Infection	1(1)	..	..	..	..	..	..	..	1
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	..	..	..	..	..	..	7	..	7
Puerperal Fever	6(1)	..	1(1)	..	..	..	..	..	7
Rubella	..	6(5)	..	1	..	..	..	..	7
Salmonella Infection	..	..	..	2(1)	1(1)	..	1	..	4
Scarlet Fever	9(4)	19(11)	4	5(3)	1(1)	4	..	..	42
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	..	..	..	..	..
Trachoma	..	..	..	..	..	..	3	..	3
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	38(25)	20(12)	25(8)	2(2)	9(7)	5(3)	3	..	102
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

the country. Dr. Cameron said that soil, as a potential carrier of disease, was a prohibited import under quarantine regulations. For example, all imported plants had to be free of soil, and any soil carried as ballast by ships entering Australian ports had to be dumped at sea. Soil could introduce such human diseases as hookworm and dysentery, the animal scourges foot and mouth disease and anthrax, both of which could also infect humans, and plant pest or disease organisms such as the cysts of nematodes, the minute eelworms which were now recognized to be a major cause of crop losses. Weeds could also be introduced with soil. Dr. Cameron said that the plant quarantine regulations provided for strict control or prohibition of seed importations as a means of avoiding the introduction of seed-borne plant diseases. The toys impounded include inflatable types containing soil ballast to keep them upright in water. Seeds had been placed in other toys to cause them to rattle. Overseas manufacturers of such toys were being advised of Australian quarantine laws.

B.S.I. Laboratory Furniture and Fittings Recommendations.

The recommendations of the British Standards Institute relating to laboratory furniture and fittings have been published as B.S. 3202; this is now available from the Standards Association of Australia, Science House, Gloucester Street, Sydney. The foreword states that the purpose of the publication is to deal with problems that are specific to laboratories. Although the recommendations represent, as far as can be ascertained, the best modern practice, they should be treated only as a guide. They do not attempt to deal with many problems that may arise in connexion with one or other type of specialized laboratory, or with general problems of design and construction which laboratories share with buildings intended for other purposes. The recommendations refer primarily to laboratories of a fairly advanced type, such as are required, for example, in universities, technical colleges, research institutions and industrial organizations. Sections deal with general recommendations on laboratory design, laboratory benches, fume extraction and laboratory services. Illustrations of typical recommended equipment are included.

A Home for the Care of Mothers and Babies at Canberra.

The Commonwealth Minister for Health, Dr. D. A. Cameron, has recently announced that the Commonwealth Government has agreed to the construction and equipment of a home for the post-natal care of mothers and babies at Canberra. The provision of the home is expected to cost £39,500. The home is to be called the Queen Elizabeth II Coronation Home for Post-Natal Care. It has decided that, subject to the concurrence of the trustees of the Queen Elizabeth II Coronation Trust Fund in the A.C.T., the money standing to the credit of the fund should be a contribution towards some particular portion or facility to be provided in the home, and should be so identified.

Dr. Cameron said that provision for the erection of such a building on a site already allocated had been made by the National Capital Development Commission in the particulars of expenditure for 1960-1961 submitted to the Minister for the Interior. Accommodation would be provided in the initial stages for two mothers with their babies, with six additional cots for babies, to meet present needs. The home would be so constructed, however, as to be capable of expansion. The Government had been concerned for some time by the fact that, whereas the cost of establishing the home would be almost £40,000, only £5215 was available towards the cost from the A.C.T. Queen Elizabeth Coronation Fund appeal. An initial sum of £2000 had been granted by the Commonwealth, and this had been brought to £5215 by public subscriptions and bank interest. The Government recognized, however, that there was a need for such a home in Canberra, and it was thought desirable that the National Capital should take suitable steps to commemorate the coronation of Her Majesty the Queen. It was expected that at the outset the home would require an annual subsidy of some £4000, after payment of fees. This could be expected to increase as the home was enlarged. However, the Government had agreed to meet this deficit.

Dr. Cameron said the home would be built at Civic Centre. The National Capital Development Commission, which had already prepared tentative sketch plans, would be asked to begin construction as soon as possible.

Deaths.

THE following deaths have been announced:

WILLIAMS.—Cyril Henry Williams, on June 4, 1960, at Brisbane.

SCOTT.—John Alexander Scott, on June 6, 1960, at Malvern, Victoria.

Diary for the Month.

JUNE 21.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JUNE 22.—Victorian Branch, B.M.A.: Branch Council Meeting.
JUNE 23.—New South Wales Branch, B.M.A.: Clinical Meeting.
JUNE 24.—Queensland Branch, B.M.A.: Council Meeting.
JUNE 28.—Tasmanian Branch, B.M.A.: Southern Subdivision.
JUNE 28.—New South Wales Branch, B.M.A.: Hospitals Committee.
JUNE 29.—South Australian Branch, B.M.A.: Annual Meeting.
JUNE 30.—Tasmanian Branch, B.M.A.: Northern Subdivision.
JUNE 30.—New South Wales Branch, B.M.A.: Branch Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

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